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A SIMPLIFIED AUTOMATIC BLOOD PRESSURE RECORDER

FRANK W. HARTMAN, M.D.* and VIVIAN G. BEHRMANN, Ph.D.**

Over the past half century numerous methods for the recording of systolic and/or diastolic blood pressure have been put forth. In sphygmomanometry the technics of recording have been plethysmographic, mechanical, optical, audiometric and the direct intra-arterial method in man, which has become the accepted standard of accuracy. None of these elaborate devices have come into general clinical usage, because of size, complexities, and cost.

There is a clinical demand for automatic blood pressure recording in the study of hypertension, in physical fitness testing, and in anesthesia. The apparatus presented herein should meet this need since it is a compact, portable, mechanical arrangement which will record systolic and diastolic pressures intermittently over long periods without discomfort to the patient. The design and method of operation are described. Records of blood pressure at rest, and as affected by exercise and amyl nitrite inhalation illustrate the usefulness of the instrument. The clinical evaluation is based upon a consideration of accuracy and practicality.

Equipment, designed for continuous recording of both systolic and diastolic blood pressure, has been presented for clinical use by Erlanger[1] (1904), Fantus[2] (1917), Kolls[3] (1920), Blankenhorn[4] (1921), Slocum and his associates[5,6] (1941, 1942), as well as by Groedel and Miller[7] (1943). The modified Erlanger sphygmomanometer, designed by Fantus and the double cuff continuous method of Kolls have introduced concepts which have proved useful in the practical application of mechanical recording.

In planning this device, the literature and the experience of others were carefully studied. In fact, features from the contributions of Fantus, Slocum, and Kolls have been incorporated in the design of this blood pressure recorder. It is a mechanical sphygmomanometer which employs two cuffs. The upper cuff serves in the production of a calibration curve while the lower one creates a pressure wave, which starts at systole and shows a pulsatile rise to a pulseless maximum at diastole.

A diagram of the blood pressure recorder is shown in figure 1. A conventional pressure cuff, applied to the upper arm, is automatically inflated and deflated at desired intervals (1-3 min.). Air pressure is admitted and released through electromagnetic valves actuated by a synchronous motor. A Haydon clock operates a pair of micro-switches so as to allow the current to flow through the solenoids of the electromagnetic valves. When this occurs in valve 1, the iron core of the valve plunger is raised and the compressed air fills the cuff; exerts its pressure

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on the small heavy walled sylphon bellows, equipped with an ink writer; and registers on the Tycos aneroid gauge in mm. Hg. When valve 2 is actuated (1-3 minutes after valve 1) the same operation causes the air pressure to escape, thus allowing deflation. In the period between inflation and deflation, an adjustable slow-bleeding valve at the upper cuff allows a gradual decline from the peak pressure, well above systolic to a point below diastolic pressure at deflation. This gradual decrease in pressure is registered as a calibration both on the Tycos dial and recorded on kymograph paper, graduated in 5 mm. intervals from 0-300 mm. Hg. through the pen actuated by the sylphon bellows. A second cuff, placed on the forearm, and inflated to a fixed pressure (three-tenths of the diastolic pressure) is connected to another Tycos gauge and a more sensitive sylphon bellows, whose pen swings from the same axis as the other pen.

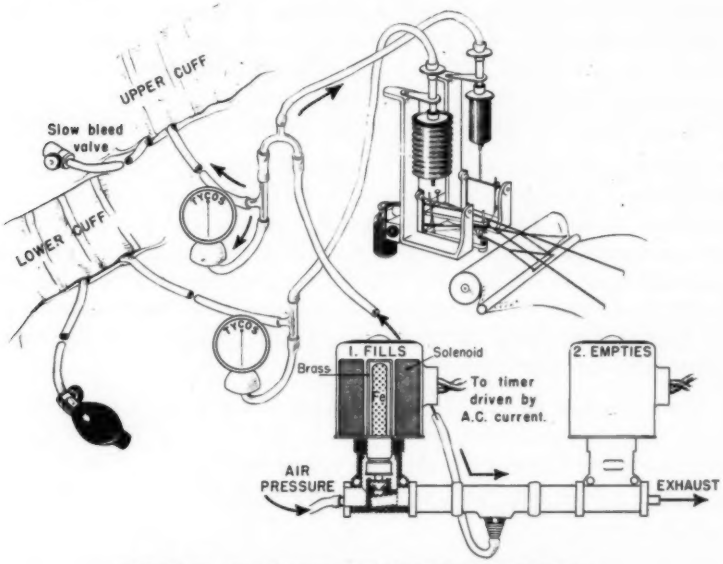

DIAGRAM OF BLOOD PRESSURE RECORDER.

Fig. 1

A pantograph of a typical blood pressure record is shown in figure 2. The tracing is read from right to left. The slow-bleed valve on the upper cuff allows a gradual decline from the peak pressure of 212 mm. Hg. to 43 mm. Hg. The ink writer from the heavy walled sylphon bellows records this pressure decrease as a calibration curve. Pressure fluctuations in the lower cuff create a characteristic curve during each interval, namely: a plateau, a sharp rise (systolic) to a peak level (diastolic) with subsequent decline, accentuated by deflation. This curve is transcribed on the paper through pressure variations within the thin walled sylphon bellows. Systolic and diastolic pressures are determined respectively by projecting (1) the break in the plateau and (2) the inception of the maximum level onto the descending calibration curve.

Blood pressure tracings on two normal individuals at rest are depicted in figures 3 and 4. One tracing shows an almost constant finding of 108/75 in six successive measurements (Fig. 3) whereas the other record indicates some degree of fluctuation (Fig. 4).

Examples of blood pressure recording in hypotensive and hypertensive states are illustrated in figures 5, 6, and 7. As the subject rests and becomes more relaxed with the device in operation, one observes a gradual decline in blood pressure in the hypotensive (Fig. 5) and in one of the hypertensive individuals (Fig. 6). The tracing of hypertension shown in figure 7 indicates a more variable systolic pressure and a more or less fixed diastolic pressure which does not respond to rest.

Continuous blood pressure recorded prior to and immediately following mild exercise is shown in figure 8. One may observe the steady blood pressure level maintained for several minutes before the bout of exercise. During the knee-bending at the rate of 30 times per minute for a two minute period, the tracing is vitiated due to movement. On cessation of exercise, elevation of blood pressure and an increased pulse pressure is immediately recorded. As subsequent determinations are made, a gradual return toward the pre-exercise level is visible.

The application of this recorder in the study of drugs which affect blood pressure is demonstrated in figure 9. Inhalation of the vasodilator, amyl nitrite creates an immediate 40 mm. Hg. decline in systolic pressure and a 24 mm. Hg. fall in diastolic pressure. It is interesting to note that the pressure in the lower cuff falls markedly as an immediate after effect of the inhalation. As the blood pressure is gradually restored, there is a concomitant increase in the height of the lower cuff curve. After eight minutes, both the blood pressure and the curve are comparable to the pre-administration findings.

The blood pressure records shown in figures 3-9 were checked at frequent intervals by the auscultatory method. In several hundred observations, agreement was within ± 3 mm. Hg., which should be adequate for clinical purposes.

The intra-arterial method (Sanborn) was recorded simultaneously with the mechanical sphygmomanometer in a few trial runs. Intra-arterial pressures from the left arm agreed with the mechanical tracing from the right arm within 5-10 mm. Hg. When the direct method was applied to the arm, used in the double cuff method, the intra-arterial recording was obliterated during the pre-systolic period as one would expect. It is difficult, then, to record the pressure from the same point in the arterial tree simultaneously with both methods. Perhaps, one should not expect to obtain the same values with the auscultatory or cuff method as with intra-arterial pressure measurement.

Intermittent recording of blood pressure over prolonged periods is the feature which should make this instrument clinically useful. Relative changes in arterial pressure, as affected by rest, narcosis, anesthesia, drugs, or exercise, may be made a part of the patient's permanent record.

SUMMARY

A simplified automatic blood pressure recorder is described. The mechanism

and operation of this double cuff sphygmomanometer is set forth. Typical recordings of blood pressure illustrate the method. Simplicity, portability, low cost and permanent recording should make clinical application feasible.

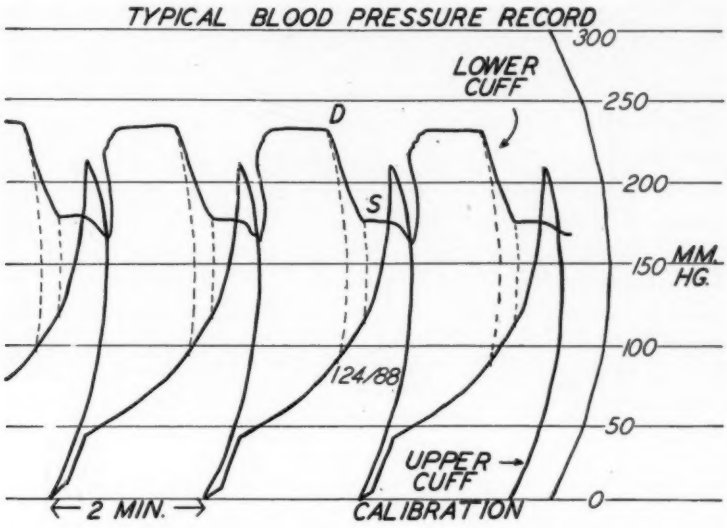

Fig. 2—In Figs. 2-9 the following terms are used: (Upper cuff) indicates the calibration tracing in mm. Hg; (Lower cuff) indicates the tracing made through pressure fluctuations in the lower cuff during each interval; (S) represents systole; (D) represents diastole; longitudinal divisions on the paper represent one minute; and (2 min.) indicates the duration of each blood pressure determination. Read from right to left.

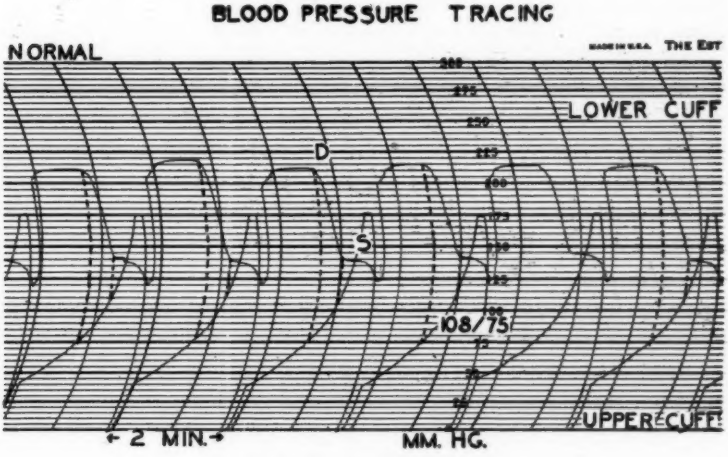

Fig. 3—(See Fig. 2 for legend)

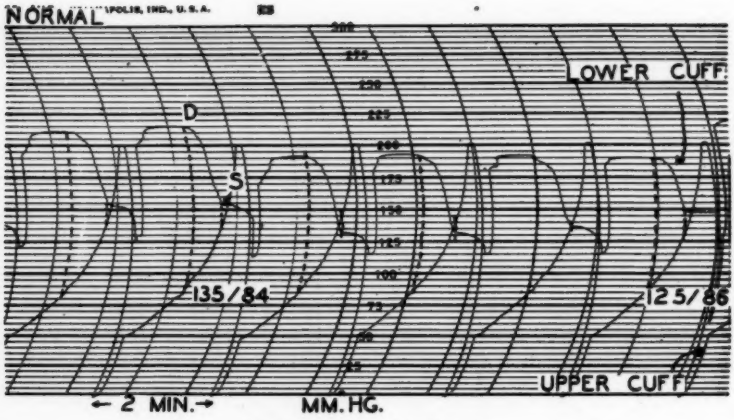

Fig. 4—(See Fig. 2 for legend)

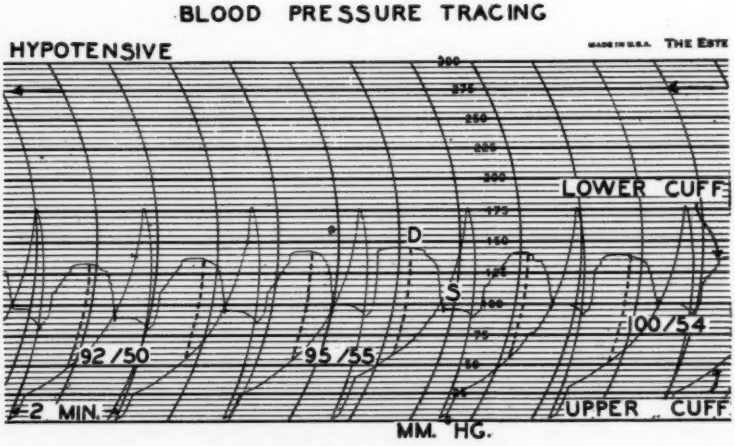

Fig. 5—(See Fig. 2 for legend)

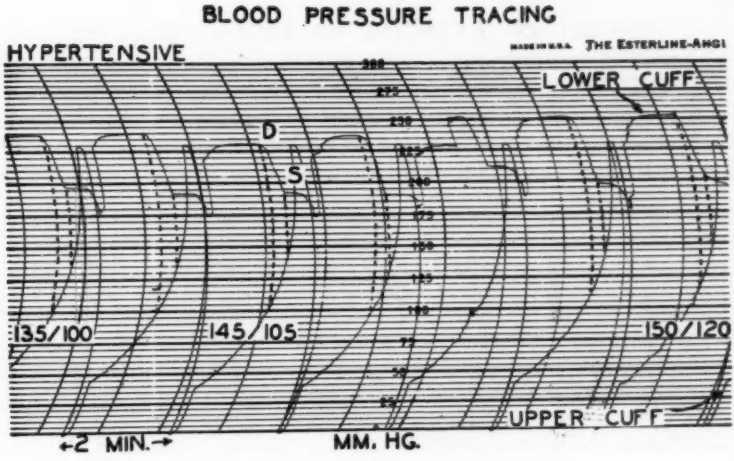

Fig. 6—(See Fig. 2 for legend)

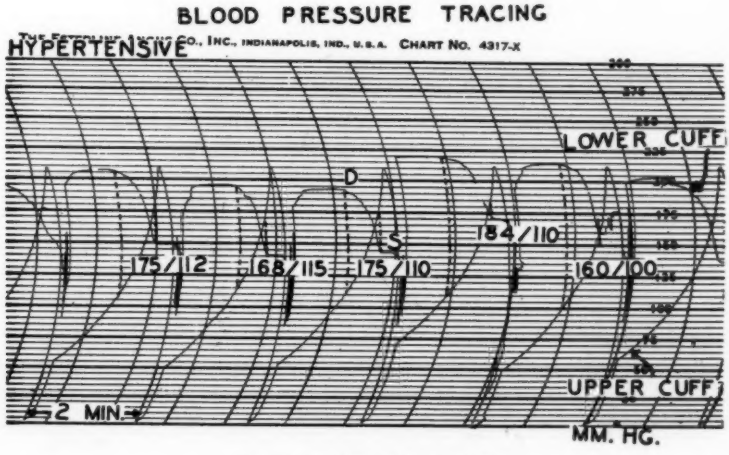

Fig. 7—(See Fig. 2 for legend)

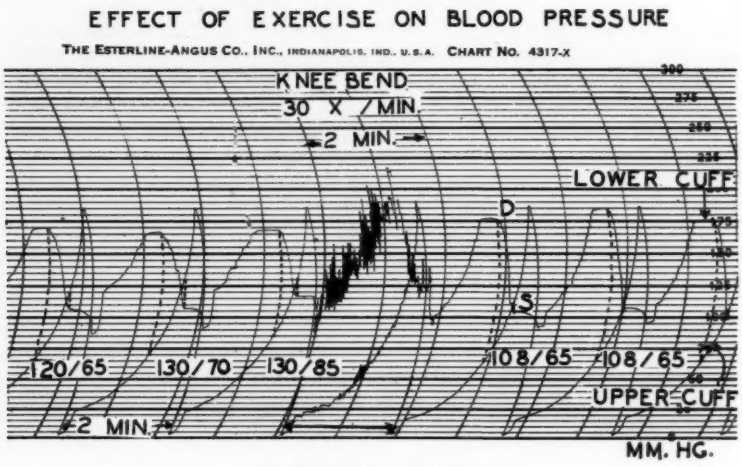

Fig. 8—(See Fig. 2 for legend)

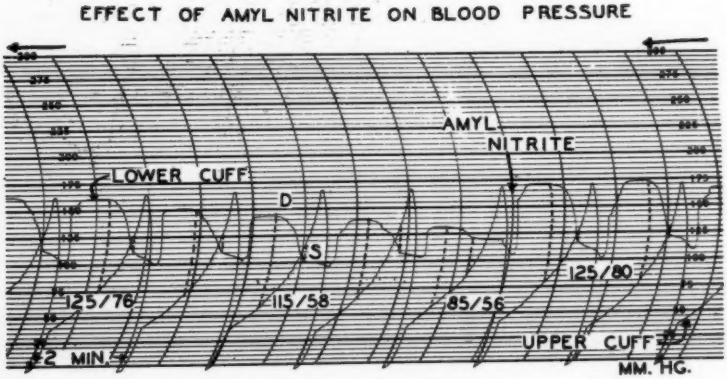

Fig. 9—(See Fig. 2 for legend)

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THE IMPRINT TECHNIQUE AS AN ADJUNCT IN THE STUDY OF LYMPH NODE NEOPLASMS

J. C. Sieracki, M.D.*

The diseases affecting lymph nodes are many and varied, their diagnosis by routine histologic methods is often difficult. Added to this is the field of the so-called "malignant lymphomas" are the marked variations in classifications schemes which have made this subject unnecessarily confusing. It is interesting that in spite of the marked variability in taxonomy of these diseases almost all workers in the field agree that there are but three major cell groups affected. The chasm often seen between the diagnoses of some clinical pathologists and anatomical pathologists on the same individual is indeed alarming and disturbing. Further, the variegated neoplastic diseases of lymph nodes often assert their presence in many different fields of medicine, e.g., the abdominal mass often palpated in a surgical clinic; the cutaneous manifestations in dermatological practices; the generalized or focal lymphadenopathy in any practice or clinic, etc., and as a result many clinical and distributional classifications have added to the problem of diagnosis.

The classification based on cytological type has been shown to be remarkable constant and appears to correlate with clinical pictures as well.[1] The probability of the development of a leukemia can also be roughly prognosticated when the diagnosis according to cell type is made.[2]

However, in much of the routine material submitted to the pathologist the cytologic details needed for such a diagnosis is not available. The same problem is faced when he is given only sectioned material from a bone marrow biopsy to diagnose various blood dyscrasias. So too, is the solution similar in each case. While the use of smears or imprints is commonplace in hematology, it is not as often used in helping the pathologist in the study of lymph node neoplasms. The purpose of this paper is to show how the use of lymph node imprints or smears can be used as an effective supplementary aid in the diagnosis, by cell type, of the lymphomas.

The term lymphoma (lymphosarcoma, lymphoblastoma) is the diagnosis used for all presently considered neoplastic diseases of lymph nodes when the only tissue examined is a lymph node. If bone marrow and/or blood shows the presence of neoplastic elements with or without a tumor of the lymph nodes the diagnosis is then termed lymphocytic leukemia (or if reticulum cells are involved primarily, leukemic reticulo-endotheliosis or monocytic leukemia of Shilling).

BRIEF HISTORICAL BACKGROUND:

The use of the imprint or smear technic on tissues other than blood or bone marrow is not new. Paul Ehrlich[3] used this technic in studying hepatic tissue in

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1883. Much of the emphasis on the use of the freshly air dried imprints or smears in the study of lymph nodes and other hematopoietic tissues in this country can be credited to Downey and Kirshbaum[4], later Sundberg and Downey[5]. The role of the lymphoid cells in mycosis fungoides was aptly demonstrated by Berman[6] using this method. Sundberg[7] offered the most plausible explanation of lymphocytogenesis in man by this technique. The important difference of nuclear structure vs. nuclear pattern of lymphoid cells was elucidated by Jones[8]. Rebuck[9] was one of the first to show morphological resemblance of pathologic reticulum cells to the Reed-Sternberg cells seen in Hodgkin's disease. The latter work also showed by the effective aid of imprints, the similarity of the cells of the reticulum cell type of lymphoma and the so-called Hodgkin's sarcoma. This was confirmed, by the same means, by Berman[2] who also aided our understanding of the relation of the "lymphomas" to leukemia.

The dry imprint or smear technique, valuable as it may be, should never be relied upon solely for pathologic diagnoses in tumors in lymph nodes. It is restated at this point that its virtue is as a supplementary procedure to the routine microscopic examination of sectioned material of the lymphoid organs. To this must be added, where feasible, the history, physical examination and laboratory data for complete evaluation of the patient.

CLASSIFICATION:

As mentioned previously, the classification in lymphoid neoplasms is indeed confusing and complex with many varied schemes offered [1, 2, 10, 11, 12]. Most oncologists have agreed that the most constant morphological factor is the cell type. Since there are but three basic morphologically distinguishable groups of cells, theoretically there should be but three groups of neoplasms. However, the occurrence of mixtures of cells plus physiological variants and varied clinical pictures requires a more complex scheme. The following is offered as a possible classification of these diseases.

HISTO-CYTOLOGICAL CLASSIFICATION OF NEOPLASTIC LESIONS OF LYMPH NODES

(Malignant Lymphomas)

I. Reticulum Cell Type
(Rarely Leukemic)

II. Lymphoblastic Cell Type
(38-50% Leukemic)

III. Lymphocytic Type
(48-80% Leukemic)

Mixed

I. Hodgkin's Type
   A. Sarcoma (Morph. indistinguishable from Reticulum cell type)
   B. Granuloma
   C. Para Granuloma

II. Giant Follicular Lymphoma.

No originality for the above table is claimed by the author. However, it is believed to be based on most of the available knowledge to date.

The predominant cell type will determine the position in the so-called "pure" tumors of lymph nodes as represented to the left of the midline above. The incidence of leukemia shows a progressive increase as we go down the list. Also the size of the cells decrease in the same order, viz., reticulum cell > lymphoblast > lymphocyte (in addition, this in general terms, parallels normal lymphocytogenesis). It is believed that the smaller cells are less likely to be trapped in the tissues and therefore gain access to the blood stream in large numbers. The reticulum cell type and all members of the mixed group are rarely leukemic. The lymphoblastic group often is accompanied by leukemia, up to 50% according to Berman[2]. The older term leukosarcoma was often used to designate this complication. Lymphocytic cell type usually shows a leukemic picture in the course of the disease. In Jackson and Parker's[10] series of 303 cases of lymphocytoma and lymphoblastoma 263 patients had a manifest leukemia.

In the mixed types the order listed roughly parallels the life span or prognosis. Though there are occassional exceptions, the sarcomatous variety of Hodgkin's disease is invariably fatal in two years, in one series[10]; the granulomatous generally leads to death in one to three years. The paragranulomatous variety has a comparatively good prognosis, 55% being alive and symptom free for five years or more, while 18% of the same series survived a fifteen year period.

In the Hodgkin's group, in the table, the interesting progression of the disease from the paragranuloma to the granuloma and the latter to the sarcoma is an accepted view. The reverse order is never seen. The giant-follicular lymphoma is an intermediate form of tumor.

METHOD:

The unfixed specimen, immediately following its removal, is sectioned with a sharp knife. The cut surface is touched lightly against a scrupulously cleaned slide. This is air-dried rapidly and subsequently treated as a bone marrow aspirate or peripheral blood smear, usually being stained by some Romanowsky dye. The procedure commonly used at the Henry Ford Hospital is the Strumia modification of the May-Gruenwald-Giemsa stain.[13]

If the dried imprints can not be used immediately they should be fixed in absolute methyl alcohol. Also, when the time element is important, they can be satisfactorily stained in 40 secs. using a solution of 0.5% Toluidine Blue 0 in 20% alcohol, to which 1% acetic acid is added.

Best cytological detail is retained if the imprint is made within two hours of its removal from the body. Immediately following the contact or touch preparation, the lymph node is then placed in a fixative and routine 5μ sections are cut and stained with hematoxylin and eosin or other useful dyes.

Histological examination will ascertain whether or not a neoplastic lesion is present because this is the *only* method of studying the topography of the lesions, invasiveness, loss of normal architecture, etc.

OBSERVATIONS:

The simultaneous study of lymph node sections and imprints requires some working knowledge of lymphocytogenesis. It is truly beyond the scope of this paper to either discuss the many theories in detail or present numerous transitional phases of this process, according to any one school of thought. Bloom[16] and Sundberg[7] adequately discuss these theories. In lymph nodes the developmental maturation series, in general terms, of reticulum cell ⟶ lymphoblast ⟶ mature lymphocyte would cause little alarm in most circles. This method of production of lymphocytes through a series of *different* cell types is termed *heteroplastic lymphocytogenesis*. In tumors of lymph nodes there is chiefly a homeoplastic process, most of the mitoses found in *one* cell type. The characteristics of the individual cells in imprints and sections will be described briefly in a simplified form.

In figure 1 we have an imprint of a relatively normal lymph node. The arrow points to a reticulum cell which has a typical nuclear chromatin pattern, a nucleolus and a large amount of cytoplasm. The latter is subject to considerable variation. The average size of these cells is 15-25μ. The irregular network of variable-sized nuclear particles making up the chromatin, along with the irregular, clear interstices (parachromatin) make this an easy cell to identify. Its counterpart in sectioned material is shown in figure 3, which is taken from a reticulum cell type of lymphoma. In figure 4, is an imprint of the same node. Though these are neoplastic cells, the basic similarity to the same type cell in figure 1, is apparent.

The large cell in figure 2 represents a lymphoblast as seen in normal node imprint. The average size of these cells is 10-18μ, they often have a narrow band of cytoplasm. The nucleus is round or oval and their chromatin pattern is finely stippled or "reticulated." They may have 0-5 nucleoli. Its malignant counterpart is illustrated in a section of a lymphoblastic lymphoma (two of the marked cells are in mitosis) figure 5. The imprint in figure 6 is taken from the same node and the large marked cells are neoplastic lymphoblasts. As can be seen from figure 5 the predominant cell type is difficult to evaluate in sections.

The mature lymphocytes are familiar, to all who have studied air dried blood smears and hardly need any description here. Figure 7 is a section from a lymphocytic type of lymphoma, while figure 8 is an imprint of the same node.

Hodgkin's sarcoma is illustrated in figure 9 with its prominent tumor giant cell (Reed-Sternberg, Greenfield) and pathologic reticulum cells. The marked cell in figure 10 is a smaller giant cell, as shown in an imprint from the same lymph node.

DISCUSSION:

The morphological diagnosis of neoplastic lymph node lesions using histologic and cytologic methods should be more accurate than study of sectioned material alone. The diagnoses of the clinical pathologist and anatomical pathologist on a

given patient can be made on the observation of the same cells under similar conditions. The use of imprints as a supplementary aid offers to the surgical pathologist neoplastic cells of a given lymph node in a state which allows more complete examination of cellular morphology. It is estimated that cells shrink 11-20% following fixation in 10% formaldehyde, hence the marked differences in cell sizes in the same cells when compared with the imprint. Since there are three cell types in lymphomas, the usual histological grading of degrees of differentiation (as done in carcinomas) is not possible and the imprint study offers a more accurate definition of the predominant cell type.

So too, metastatic tumors of lymph nodes occasionally offer a difficult problem in deciding whether the lesion is primary or secondary. This is especially true of transitional cell carcinomas and unpigmented melanomas.[15] If the large marked cells in figure 11 are compared with those of figure 3 they appear quite similar; yet the latter is a section from a reticulum cell lymphoma while figure 11 is a section of a lymph node invaded by metastatic transitional cell carcinoma. On imprint from the same node figure 12, the foreign neoplastic cell can be readily differentiated. The misinterpretation of a lymphoma (especially reticulum cell type) as a carcinoma can also be avoided in some cases, if this technique is used.

In many instances the surgeon requires a diagnosis on some lymphatic tissue while he is operating. Good rapid fresh frozen sections of this material are at times difficult to obtain due to the soft consistency of lymphoid tissue. The lymphoma represented in figures 3 and 4 was diagnosed using both sections and imprints when the surgeon was confronted with a retroperitoneal mass near the pancreas. His first biopsy was histologically pancreatic tissue with no evidence of malignancy. The second biopsy of the mass on frozen section appeared neoplastic. Imprints of this mass showed sheets of reticulum cells.

In infections mononucleosis, histological distinction from a lymphoma can be almost impossible. The imprints will show leukocytoid lymphocytes in abundance and a heteroplastic rather than homeoplastic lymphocytogenesis.

Mycosis fungoides[6] often a difficult histologic problem, as well as Hodgkin's Disease[15] with cutaneous involvement can be resolved in some instances by the aid of imprints of skin lesions.

The use of cellular preparations obtained by needle biopsy of lymph nodes, or imprints without histological examination is dangerous. In giant follicular lymphoma the predominant cell type may be any of three major groups and a grave error in subsequent prognosis could be made. Likewise imprints from nodes in Boeck's Sarcoid and severe inflammations, may show hyperplasia of one cell type while Hodgkin's Disease may give varied types.

Further advantages of the method not mentioned above are its relative simplicity and its value as a second diagnostic approach on the same biopsy material. It is inexpensive and does not interfere with subsequent use of the excised tissue for histologic study.

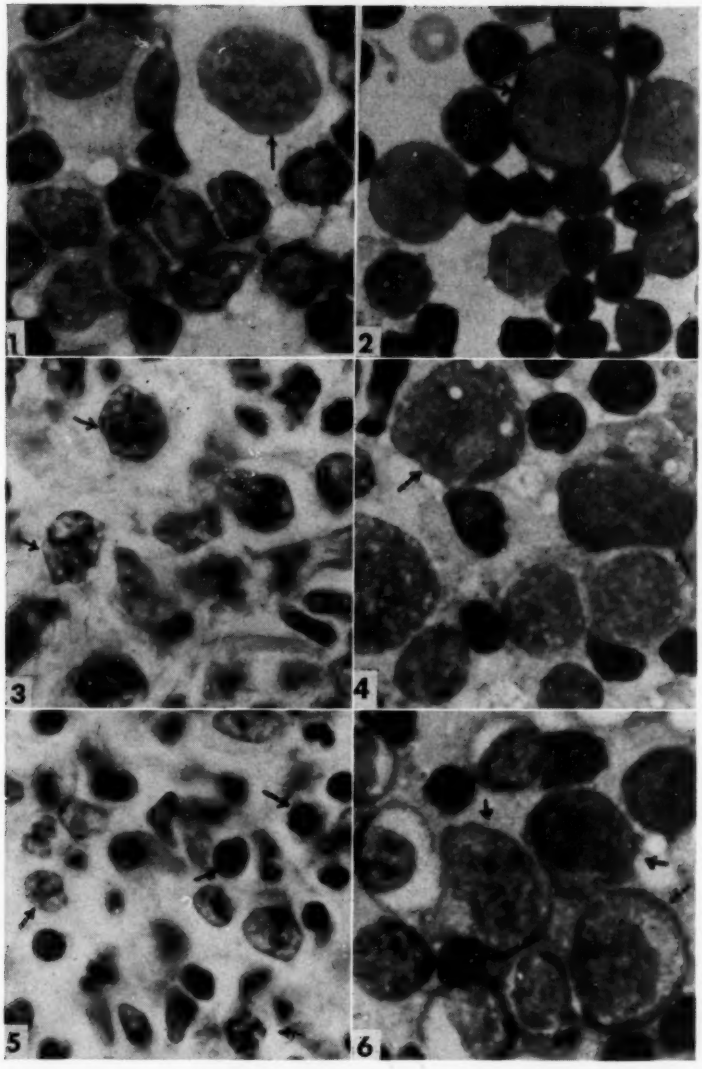

PLATE I

All figures at same magnification 1150 x.
All imprints stained with May-Grunewald-Giemsa stain.
All sections stained with H & E.

Fig. 1—Imprint of normal lymph node. Large cell (↑) in upper right is normal reticulum cell.

Fig. 2—Imprint of normal lymph node. Large cell (↑) in upper center is a lymphoblast and smaller darker cells in center are lymphocytes.

Fig. 3—Section of lymph node, lymphoma, recticulum cell type. Large cells (↑) are reticulum cells.

Fig. 4—Imprint of same lymph node shown in figure 3. Large cells (↟) with vacuoles are reticulum cells.

Fig. 5—Section of lymph node, lymphoma, lymphoblastic cell type. Marked cells (↟) are lymphoblasts, two of which are in mitoses.

Fig. 6—Imprint of same lymph node shown in figure 5. Marked cells (↟) are lymphoblasts.

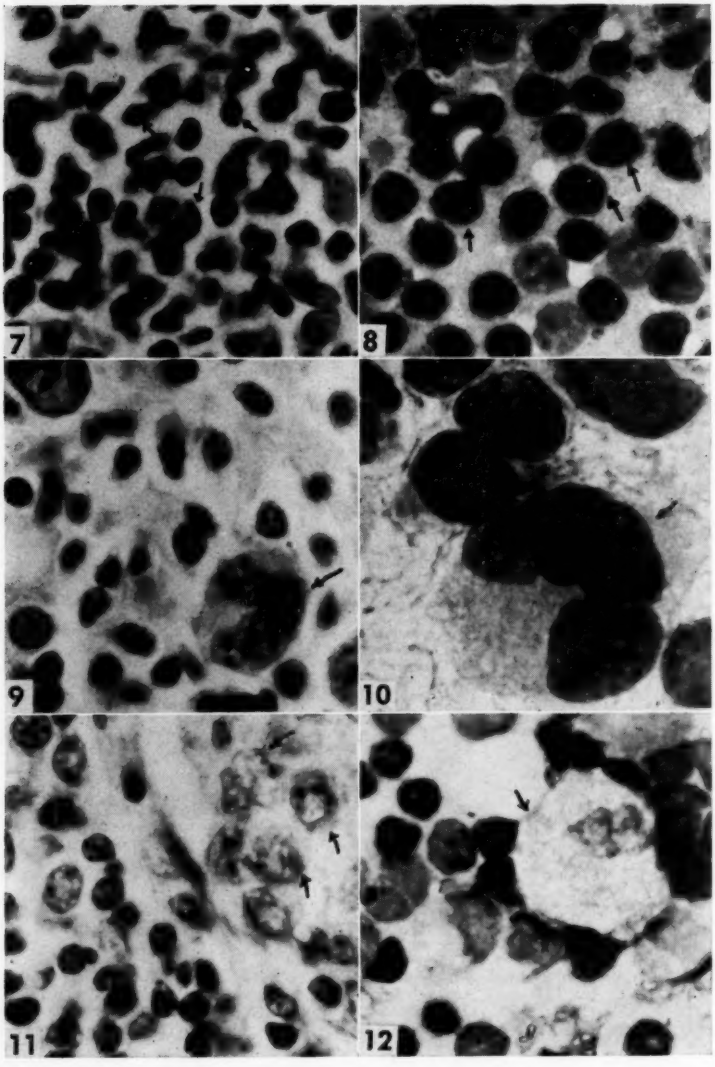

PLATE II

Fig. 7—Section of lymph node, lymphoma, lymphocytic cell type. Numerous small dark cells are lymphocytes, some of these are marked (↑).

Fig. 8—Imprint of same lymph node shown in figure 7, showing numerous lymphocytes.

Fig. 9—Section of lymph node from Hodgkin's Sarcoma showing prominent bizarre giant cell.

Fig. 10—Imprint of same lymph node showing in figure 9, showing a similar but smaller tumor giant cell.

Fig. 11—Section of cervical lymph node showing metastatic cells from a transitional cell carcinoma of nasopharynx. Note similarity of marked cells (↑) to reticulum cells (fig. 3).

Fig. 12—Imprint of same node shown in figure 11. Marked cell (↑) is metastatic carcinoma cell.

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SEGMENTAL ILEITIS DUE TO SCHISTOSOMIASIS MANSONI

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This material is being presented for several reasons. Schistosomiasis is an unusual disease in this part of the world and opportunities to study its pathology are rare. Furthermore the particular complication exhibited by this patient is uncommon for schistosomiasis. Lastly the clinical and pathological features provide a situation of interest from a standpoint of differential diagnoses. No effort will be made to review the life cycle of the parasite since it is ably presented in numerous sources.[1]

The patient K.M. is a 28 year old Arabian who came to live in this country fairly recently. He was seen for the first time at Henry Ford Hospital on December 18, 1952 with the chief complaint of abdominal pain. His pain had been present for about four weeks and appeared worse after eating. There was slight nausea but no vomiting. Physical examination showed no unusual findings. Laboratory studies disclosed 4 + cephalin and thymol flocculations, but other routine tests were normal. Biliary drainage produced normal appearing bile. However, microscopic examination disclosed ova of the *Schistosoma mansoni* type. Stool examinations were carried out and the ova seen again, together with *Trichuris trichiura*. It was decided that the picture was due to schistosomiasis and fuadin therapy administered.

During subsequent visits in the outpatient department a 2-3 cm. mass was noted in the right lower abdomen and a smaller mass on the left side. The liver was slightly enlarged and an x-ray showed a suggestion of fluid levels in the small bowel suggesting early intestinal obstruction.

The abdominal cramps recurred and were especially severe after meals. Again there was no vomiting and no diarrhea. Some weight loss had occurred. Because of the symptoms he was readmitted January 1, 1953. There was a tender mass to the right of the umbilicus. Laboratory studies were normal except for mild anemia. X-ray studies showed multiple polypoid defects of the terminal ileum.

He was again followed as an out patient, but because of persistence of pain and beginning abdominal distention it was decided to readmit the patient for exploration of the abdominal mass. At operation (July 27, 1953) a huge mass of enlarged mesenteric lymph nodes was encountered in the right lower abdomen, and the terminal ileum was thickened and indurated with almost complete obstruction. The terminal ileum and ascending colon were resected and bowel continuity restored. Postoperatively the patient did well. On August 10, 1953 he was seen again because of persistent epigastric pain. This was thought due to nerve involvement in the operative scar. Repeat x-ray studies showed a well functioning anastomosis.

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Examination of the specimen removed at operation showed the mesenteric nodes to be greatly enlarged and firm. There was some fusion of adjacent nodes. On section they were dense grey white with scattered yellow areas. The terminal 6 cm. of the ileum showed a partial obstruction due to an edematous, polypoid swelling of the mucosa which was sharply localized and ended abruptly at the ileocecal valve. (Fig. 1). There was only a small superficial ulceration. The bowel appeared pale and edematous rather than hemorrhagic. In many ways the gross picture similated regional enteritis.

Microscopy study showed an intense infiltration of the mucosa with macrophages and scattered about were numerous abscesses in the center of which could be seen ova of the schistosoma worms in varying stages of preservation. (Fig. 2-3.) Eggs which had been present a longer time were surrounded by macrophages and giant cells. (Fig. 4.) The inflammatory process seemed out of proportion to the relatively few ova present and produced a great increase in thickness of the mucosa and submucosa. The cellular reaction consisted predominantly of macrophages together with a few lymphocytes and eosinophiles. The muscularis of the bowel was normal but heavy infiltrations were found between the muscle layers and in the serosa. The appendix and colon showed only a hyperplasia of preexisting lymphatic tissue. Sections from the mesentery demonstrated marked fibrosis and a granulomatous reaction. The circumscribed granulomas were composed of macrophages but giant cells were not present. Frequently there was central necrosis and neutrophiles were fairly abundant. Usually wide bands of fibrosis surrounded these granulomata. They had completely replaced the lymph nodes of the mesentery. No ova could be found in these areas. Another prominent feature was the heavy accumulation of lymphocytes along the course of lymphatic channels. There was also considerable edema present. The smaller veins often showed an obliterative proliferation of their intima. (Fig. 5.) In two of the larger veins there were mature worms. (Fig. 6.) These showed a moderately thick integument and the alimentary tract contained depositions of brown pigment. Periodic acid leucofuchsin stains stained the ova in the mucosa brilliantly and were of considerable help in locating these structures.

DISCUSSION:

There are numerous important differences in the diseases caused by the three species, *hematobium*, *mansoni* and *japonicum*. Our patient contracted his infection in Arabia where both the *mansoni* and *hematobium* are found. All three are characterized by the release of ova from adult worms inhabiting venules and in all three the lesions are due to extravasation of ova into tissues. The adult worms are apparently well tolerated. In hematobium infections the adults occupy veins in the pelvis, especially those of the bladder, and most of the lesions involve the urinary tract. In japonicum and mansoni infections, the adults are found in branches of the portal system, and the intestinal tract and liver are the organs which show the most damage. The japonicum infection is more severe due to production of larger numbers of ova. Mansoni infections are usually milder and more protracted. The intestinal lesions are ordinarily encountered in the large

bowel and are characterized by ulcerated polypoid lesions. These are also more severe in japonicum infections. In both diseases the hepatic lesions are more important and most cases die due to hematemesis and cirrhosis. Excellent descriptions of the lesions encountered in mansoni infections are found in Koppisch's classic studies on the disease.[2]

Our case is unusual because of the curious segmental localization of the intestinal lesions in the terminal ileum and because of their severity which resulted in partial obstruction of the bowel. There is evidence of considerable liver damage on the basis of liver function tests but no tissues were available for study. The studies in Puerto Rico demonstrated that in minimal cases only the liver and rectum were involved and that later, the adults move to higher radicles in the portal system with involvement of mesenteric nodes and small bowel. There were no cases in their series showing such marked localization of the inflammatory polyps in the terminal ileum with resultant partial obstruction. In general lesions of the small bowel were quite minimal in character in their cases except for subperitoneal nodules. None were of any clinical significance.

The histopathological features are beautifully described and interpreted in Koppisch's work and no additional information can be added. It is remarkable how few ova were encountered in this lesion considering the bulk and intensity of the inflammatory reaction. The ova elicit an acute inflammatory process at first which is followed by the appearance of macrophages and endothelial cells. Destruction of the embryo results ordinarily and the inflammatory granuloma is encapsulated and fibrosed to form pseudo tubercles. Local sensitization of tissue to toxic factors produced either by the worms or following destruction of the eggs must play an important role in view of the marked inflammatory response. Koppisch points out that the frequent occurrence of necrosis in the center of the pseudo tuberculomas also points to the possibility of an allergic phenomenon.

One feature that is not stressed in previous descriptions is the intensity of the reaction involving lymphatics. Everywhere there is marked accumulation of lymphocytes and plasma cells in and around lymphatics again indicating a response to immunological stimuli. The smaller venules frequently showed proliferation of intima even to the point of occlusion. These are lesions which occur in other granulomatous diseases of the small bowel, especially tuberculosis and terminal ileitis. The differential points between the inflammatory picture in these conditions is clearly discussed by Rappaport[3]. It is of considerable interest that many of the features noted in this case, whose pathogenesis and etiology are known, occur in other diseases. The early changes in regional enteritis closely parallel the gross features of this case. Microscopically the same edema and inflammatory lymphangitis is seen but in regional enteritis the proliferative response consists predominately of a hyperplastic lymphadenitis in which granulomatous foci are seen. Similar endophlebitic lesions are also seen and are well illustrated in Rappaport's article. These points of similarity suggest that in regional enteritis the sensitivity or allergic reaction of tissue may play an important part in producing the inflammatory reaction.

SUMMARY

A case of Schistosoma mansoni infestation is presented which developed the unusual complication of partial intestinal obstruction necessitating resection of the terminal ileum. The pathology of the disease is briefly reviewed and attention drawn to the role of tissue sensitivity in the inflammatory reaction.

Fig. 1—Terminal ileum and caecum. The inflammatory thickening of the mucosa is sharply segmentalized to the terminal ileum.

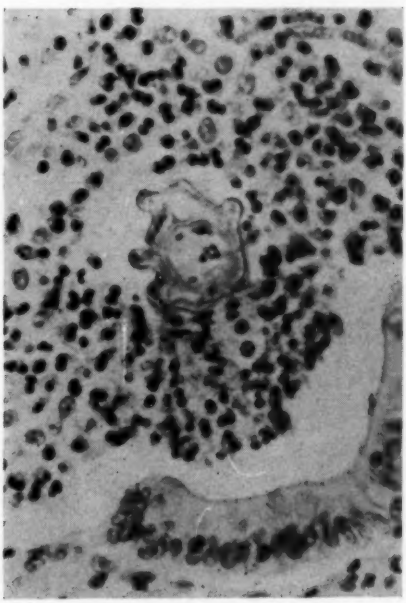

Fig. 2—Ovum being dislodged into lumen of intestinal gland.

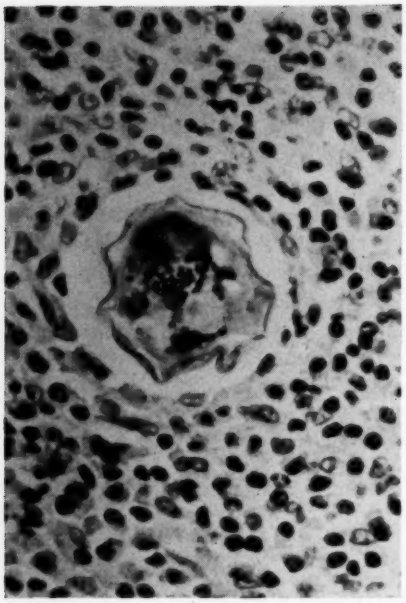

Fig. 3—Ovum with embryo still intact. There is beginning encapsulation.

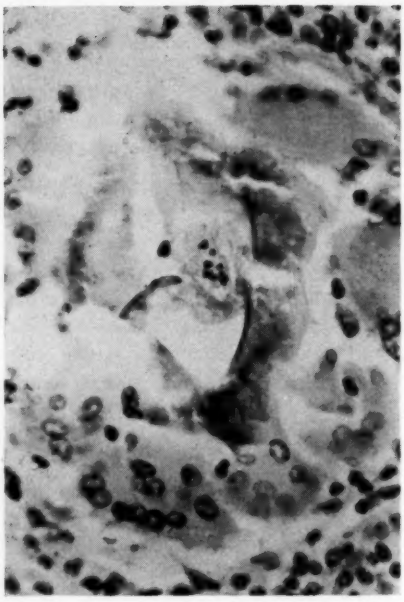

Fig. 4—Ovum undergoing destruction with foreign body giant cell formation.

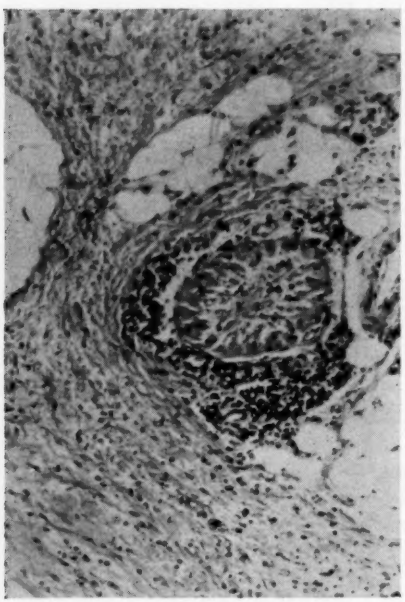

Fig. 5—Endophlebitis obliterans together with lymphangitis.

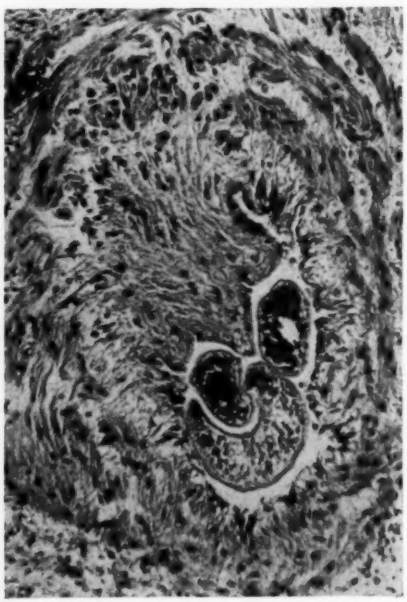

Fig. 6—Adult worms within mesenteric veins.

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INFLUENCE OF SACCHAROIDS ON BLOOD GLUCOSE VALUES

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For many years, the method most widely used in this country for the estimation of blood sugar has been that proposed by Folin and Wu[1] in 1920. With the development of other methods, it was shown that the results obtained by the Folin-Wu method were appreciably higher than the amount of fermentable carbohydrate (presumably glucose) present in human blood. These non-carbohydrate reducing compounds were called "saccharoids" by Benedict[2]. In a study of ten bloods[3], he found that the saccharoids caused the results of the Folin-Wu method to average about 22 mg. per 100 ml. too high. While studying the nature of the saccharoid fraction in human blood, Fashena[4] found that these substances produced an average error of ∓21.5 mg. per 100 ml. for the 25 blood samples studied. It was found that glutathione accounted for about 37% of the saccharoid material and that most of the remainder was related to glucuronic acid. Formerly, it had been assumed that creatinine, uric acid and ascorbic acid were important as interfering substances in the blood glucose method. The results of Fashena and Stiff[5] indicate that not more than 2-3 mg. per 100 ml. of the saccharoid error in the Folin-Wu method can be related to these substances.

Based on rather meager evidence, it has been assumed that the saccharoid content of human blood is usually 20 to 30 mg. per 100 ml. and that "the saccharoid fraction appears to be relatively constant and therefore its presence does not influence unduly the interpretation of variations in the blood sugar level."[6]

Attempts to devise methods which yield "true glucose" results have followed one of three general principles: (A) removal of glucose by fermentation by yeast, (B) modification of copper reagent to make it more specific, and (C) removal of saccharoid materials by use of special protein precipitating reagents. With the more frequent use of methods which give "true glucose" values, it has been possible to show that the saccharoid fraction is not as constant as had been assumed.

Mosenthal and Barry[7] studied two hundred consecutive blood samples and found that 38% of them contained saccharoids in excess of 30 mg. per 100 ml. In four specimens, the saccharoids were in excess of 70 mg. per 100 ml. A study was also made of the non-glucose reducing substances during 25 oral glucose tolerance tests. From the results, it is quite evident that the saccharoid fraction is not constant during this test. In four instances, the saccharoid fraction exceeded 30 mg. per 100 ml. for all samples of the test and in another 16 instances, at least one of the blood samples exceeded this limit. Thus, it is evident that blood sugar values including the saccharoid fraction may lead to erroneous interpretation of their significance.

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From the laboratory standpoint, the amount of work involved in estimating true blood glucose varies considerably with the type of method used. When methods involving modified copper reagents are used, there is very little change in the effort required; however, when either of the other two general methods is employed, there is a considerable increase in the amount of work per determination. Therefore, a study was made of the difference in results obtained when the Folin-Wu[1] and the Benedict[8,9] methods were employed on the same blood filtrate. Table I shows the difference obtained between the two methods on 204 consecutive bloods received in the laboratory.

TABLE I

Difference in Blood Sugar Values
(Folin-Wu less Benedict)

Difference mg./100 ml.	*Number of Cases*
0–10	95
11–20	78
21–30	22
31–40	4
41–50	2
51–60	2
61–70	1
Average 12.5	204

The distribution and magnitude of the saccharoid error in this data is similar to that found by Mosenthal and Barry. It must be remembered that the Benedict method gives values which have a small saccharoid error. The saccharoid error for the Benedict method, as determined before and after yeast fermentation[10], was found to amount to 4-6 mg./100 ml. of blood. In no instance was this difference found to exceed 8 mg. per 100 ml.

A study of the data shows that no relation exists between the glucose content of the blood and the magnitude of the saccharoid fraction. The largest value for saccharoids found in this series amounted to 68 mg. per 100 ml. of blood while the glucose level was only 82 mg. per 100 ml. This lack of correlation is further emphasized by the fact that four out of the five blood samples with the largest sacchraroid fraction had glucose concentrations between 70 and 100 mg. per 100 ml. Thus, these four blood samples would be considered to have normal values for blood glucose but the values by the Folin-Wu method would be in excess of the generally accepted normal values of 90 to 120 mg. per 100 ml. for this method. The failure to find many blood samples with excessive saccharoid fractions in the presence of high blood glucose may be related to the fact that only 15% of these samples contained glucose in excess of 150 mg. per 100 ml.

From the data presented, it would seem desirable to abandon the use of the Folin-Wu method of estimation of blood sugar in the clinical laboratory. It should be replaced by one of the methods known to yield true glucose values. Of these methods, it would appear that the one devised by Benedict[8,9] would find ready acceptance in most clinical laboratories.

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EXTRACTION AND PARTIAL PURIFICATION OF COXSACKIE VIRUS FROM HUMAN STOOLS WITH THE USE OF ION-EXCHANGE RESINS

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Our present methods for virus diagnosis cannot be utilized by the physician in the diagnosis of virus infection during the acute phase. Virus diagnosis now rests on the demonstration of increased antibody titer during the course of the disease and convalescence. Thus diagnosis must be made in retrospect which is of little value to the clinician. If the virus, which is known to be present during the acute stage of illness, could be extracted from the excreta and identified by means of known sera, it would place the diagnosis of certain virus infections in the category of bacteriological procedures. The purpose of this communication is to briefly describe a new laboratory tool with which to explore this phase of the diagnostic problem.

A technic for the partial purification and concentration (Ref. 1-2) of the poliomyelitis virus from central nervous system tissue using a strong base anion exchange resin (Amberlite XE-67) has been reported[2]. However, since complement fixing antibodies have not been satisfactorily demonstrated in poliomyelitis, this virus is not suitable for preliminary investigations. On the other hand, the Coxsackie virus, an agent to be considered in the differential diagnosis of poliomyelitis infection and which is also found in stools, is known to possess complement fixing properties. The agent is more suitable for the elaboration and study of this concept of early diagnosis of virus infections. For this reason the technic developed with the poliomyelitis virus was adapted to the extraction of the Coxsackie virus excreted in stools. This procedure is particularly applicable to the extraction, partial purification and concentration of virus found in the excreta of acutely ill patients. It has two major advantages: (1) its capacity for handling large volumes of crude infected material in a relatively short period of time and (2) its relative simplicity in that it can be conducted at room temperature without the use of the ultra-centrifuge.

The amount of Coxsackie virus for these studies was limited as only one positive stool was obtained out of 40 examined during the 1952 poliomyelitis season. The material obtained from this one specimen was sufficient to develop the extraction procedure given below.

METHOD

The procedure may be described briefly in three distinct steps. These are conducted at room temperature.

Step 1 involves removal of anions and cations from a 1% fecal suspension. Several liters of material can be processed in 3-4 hours or 5 gallons can be processed un-

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attended over night. The material flows through a glass column containing a mixture of two resins: one a strong base anion exchange resin in the hydroxyl form (Amberlite* IRA-410), the other a weak acid cation exchange resin in the hydrogen form (Amberlite IRC-50). IRC-50 had to be substituted in the procedure for processing of fecal material where a strong acid cation exchange resin was used for tissue material. The effluent from this column contains the virus. It flows directly into a large vessel containing the virus-adsorbing resin Amberlite XE-67.

Step 2 consists of adsorbing and eluting the virus from Amberlite XE-67. Virus adsorption to this resin is complete in 30 minutes. The resin is then recovered and the large volume of fluid discarded. The recovered resin-virus-protein complex is then treated with a small volume of 10% disodium acid phosphate which elutes the virus from the resin complex in approximately 30 minutes. This procedure leaves the bulk (88-94%) of the extraneous nitrogenous material on the resin. The purification and concentration is obtained from this step in the process.

In step 3 the excess disodium acid phosphate is removed from the virus-phosphate eluate. This can be accomplished either by dialysis against distilled water or by the use of another resin column. The resin column technic is simpler and less time consuming.

The Coxsackie virus obtained by this procedure showed a high degree of infectivity for suckling mice. However, it was first found unsatisfactory for serological work in the complement fixation test. This was due to minute traces of phosphate which proved to be anticomplementary but were subsequently removed by more thorough dialysis against distilled water. Further studies were not possible due to the lack of clinical material. However, three additional positive specimens were isolated during the 1953 poliomyelitis season, so that these investigations can now be completed.

It remains to be demonstrated that the Coxsackie virus extracted from the stools can be used as a complement fixing antigen in a serological test for an early presumptive diagnosis of Coxsackie infection. Confirmation of the disease, however, would still rest on the increase of antibody titer during convalescence. The principal value of this work is not so much its use in the early diagnosis of Coxsackie infection but rather the fact that a new "tool" has been developed which can be utilized in the search for a practical laboratory diagnosis of virus diseases in general.

*Amberlite resins were supplied through the courtesy of Rohm & Haas Co., Philadelphia, Pa.

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ELECTRON MICROSCOPY OF THE SICKLING PROCESS[a)]

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Direct visualization of corpuscular aggregation of Hb. in the sickling process was attained by modification of the Beck-Hertz technic which put the sickling process into slow action.[1] Formalin fixation at successvie times arrested the sickling process at many stages in the transformation of biconcave disc to sickle cells. Three patients with sickle cell anemia, five with sickling trait and one with Hemogloblin C anemia were so studied.

a. Hemoglobin aggregation (Paracrystallization)[2]

After 15 minutes of anoxia, peripheral aggregation of hemoglobin effected an increase in diameter of promeniscocytes over that of the normal, with focal thinning of the disc allowing passage of electrons. This thinning eventually included the entire corpuscular center. In the thinned center, even upon immediate fixation, anisotropoid angulation of central aggregates of hemoglobin was detectable. When aggregation of hemoglobin about the entire corpuscular rim was accompanied by peripheral angulation of the hemoglobin aggregates, the holly-wreath form of Sherman[3] appeared. If aggregation of hemoglobin about the corpuscular rim was incomplete at a single locus, an appearance of pseudo-rim-rupture was obtained, an uncommon finding in our material. More often there was an irregular aggregation of hemoglobin in one side of the cell (Fig. 1) so that at five hours of anoxia, the eccentric, hemoglobin-containing intracorpuscular masses presented intense peripheral spiculation suggestive of incipient crystallization or paracrystallization. This eccentric massing of the hemoglobin offering labile, but definite angulation was the key to the sickling process. The hemoglobin concentration of the normal erythrocyte is on the threshold of gelation[4] and the reduced, sickle cell hemoglobin is only one-hundredth as soluble as normal oxyhemoglobin.[5] It is therefore not surprising that the defective hemoglobin molecules of the sickle cell, when reduced, because of their increased complementariness (as proposed by Pauling and his associates[6]), are capable of entering into long chain and three-dimensional frameworks whose structural patterns are depicted in Fig. 3. Complete sickling of the sickle-cell-anemia cells eventuated in intense polar filamentation in which hemoglobin and surface ultrastructure both played important roles. Polar filamentation was inconspicuous in the sickling-trait cells and occupied an intermediate position in Hemoglobin C anemia sickling. Examination of the sickling process in the sickling-trait cells revealed that in the important, early, eccentric massing of hemoglobin aggregates, there was less spiculation or angulation of these aggregates, a finding which confirmed the ob-

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a) with the technical assistance of Miss E. A. Monaghan.

servation of Pauling and his associates[6] that such cells possess an admixture of defective and normal hemoglobin.

b. Tactoid formation, modification by surface ultrastructure.[7]

Harris[8] proposed that sickle cells are merely hemoglobin tactoids, a type of fusiform, liquid crystal, thinly veiled by the erythrocytic membrane. Electron micrographs of sickle cells which were also fully developed tactoids were next studied. (Figs. 2 and 4.) Essentially, the spindle-shape of the tactoid is the resultant of the force of surface tension which tends to make it a sphere and those of orientation potentials tending towards formation of a needle-like crystal at either end. The sites of bipolar filamentation of some of the extraneous membrane were at the very regions where the forces of crystallization were most exerted. In 1930 Graham and McCarty[9] reported a case in which all the sickle cells were needles. More often, however, sickle cells are sickle-shaped because of the distortion curvature impressed upon the otherwise fusiform tactoid by the forces inherent within the still intact portions of the surface ultrastructure.

c. Enhancement of the sickling process by injury to the surface ultrastructure.[10]

If the intermolecular forces of the surface ultrastructure could be weakened during the sickling process, not only would sickling be accelerated but hemoglobin aggregation within the corpuscle could proceed with a minimum of membranous impedance. Sydenstricker and his associates[11] in 1923 had found that bile and bile salts accelerated the sickling process. We confirmed their findings for Na glycocholate and found in addition that soap is almost equally effective as a sickling accelerator. Indeed, weakening of the surface ultrastructure with such surface active agents permitted almost free play to the opposing forces of hemoglobin orientation potentials tending to crystal formation and produced, as a result, monstrous elongated forms. When such damage to the surface ultrastructure exceeded a certain point, focal hemoglobin aggregation occurred along the entire length of the structure in the place of accentuated tactoid formation.

d. Tridimensional nature of the sickling process.[12]

Dual studies of anemia sickle cells were made as follows: Direct micrographs of individual sickle cells in all stages from promeniscocyte to completely sickled forms were made with the electron microscope. Each cell was then removed from the microscope, shadow-cast with chromium and tridimensional micrographs of the same cells were again obtained in the electron microscope for comparison. Eccentric massing of corpuscular aggregates was manifest at promeniscocytic stages previously dismissed as being merely macrocytic. Bar-like massing of the hemoglobin was confirmed tridimensionally. The bizarre outcroppings of completely sickled cells were studied from the standpoint of constituent, aggregational elevations.

SUMMARY

The electron microscopy of the sickling process was studied in the erythrocytes obtained from three patients with sickle cell anemia, five with sickling trait and

one with Hemoglobin C anemia. Massing of the hemoglobin accompanied by angulation of the hemoglobin aggregates appeared to be the primary structural abnormality. Tactoid formation together with its modification by changes in the surface ultrastructure was further investigated. The tridimensional nature of the process was observed after shadow-casting with chromium.

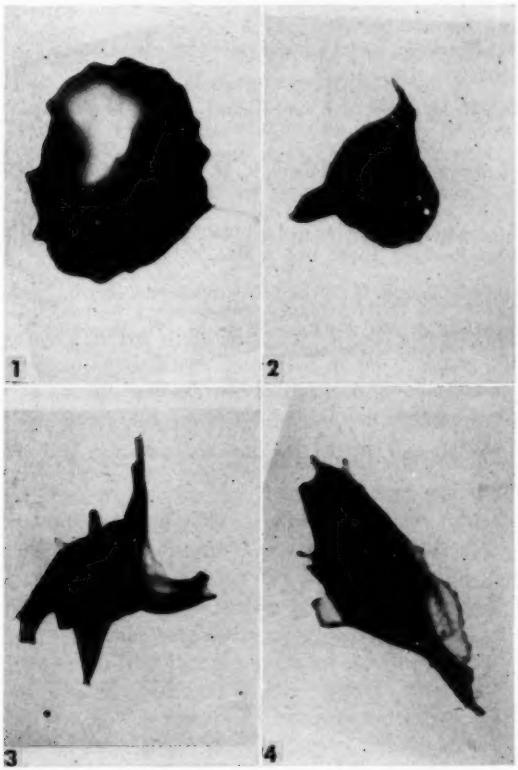

Fig. 1—Early sickling. RBC in sickle cell anemia. Eccentric massing of Hb with beginning peripheral angulation. Formalin fixation. x 5300.

Fig. 2—Early sickling. RBC in sickle cell anemia. Beginning tactoid formation. Formalin fixation. x 5300.

Fig. 3—Late sickling. RBC in sickle cell anemia. Peripheral angulation and filamentation. Formalin fixation x5300.

Fig. 4—Late sickling. RBC in sickle cell anemia. Tactoid formation with modifying surface structure. Formalin fixation x 5300.

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ROENTGENOLOGICAL APPEARANCE OF THE ABDOMEN IN ILEUS

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The diagnosis of obstruction of the small intestine is a very serious problem to the surgeon. Along with the details of the history and clinical findings, he will find that roentgenography can supply evidence which cannot be obtained in any other way: (a) the degree of bowel distention, (b) location of distended loops, (c) presence of air in the peritoneal cavity, and (d) site of obstruction.

In many instances, the radiologist who is not familiar with details of the history and clinical findings, will attempt to decide the presence of mechanical or paralytic (adynamic) ileus on the appearance of the X-rays alone. This practice should certainly be discouraged. On the other hand, the findings in the roentgenogram will prove extremely valuable to the surgeon when small bowel obstruction is suspected.

In acute obstruction of the small intestine which is certainly a surgical emergency, the patient may be too ill to be taken to the X-ray department. In such cases, the use of the mobile X-ray unit will make possible obtaining of radiograms quickly, through plain scout films of the abdomen showing the site of the obstruction, and this will cause no disturbance to the patient.

Scout films of the abdomen are obtained without contrast medium or preparation to the patient, and are usually taken in emergency cases, often at a bedside. Joseph Lavitin[1] found the scout film a significant aid in substantiating or establishing diagnosis in some conditions. In perforated ulcer, an antero-posterior view in lateral decubitus position with the left side down is desirable and may show free air in the abdomen, when the upright view fails. It is also much easier to take the film with the patient in the decubitus position when he is too ill to have a radiogram taken in the upright position. In non-opaque ureteral stones, the abdominal findings are important in the negative sense, in that no abnormal dilatation may be present. A stone on the right side may make differentiation between ureteral colic and appendicitis difficult. Such a stone may block the ureter and the characteristic signs of blood in the urine may be absent. An intravenous pyelogram may be made without disturbing the patient. If necessary, the films may be taken at bedside with a stationary grid. Spasm of the kidney pelvis may prevent excretion of the dye and density of the kidney on the side will appear increased.

During the first 24 to 48 hours after abdominal surgery, gas filled loops of bowel must not be interpreted as evidence of ileus. This has been observed to persist as long as two weeks.

In the normal subject, excluding infants and older patients, gas is not usually found in the small intestine, except for small quantities in the duodenum and

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occasionally in the terminal ileum. When the individual is unable to belch, he may swallow enough air to fill a number of loops of small intestine. However, the width of the lumen so outlined does not exceed 2.5 to 3.0 centimeters. Gas distended loops of small bowel with a fluid level are indicative of ileus (Fig. 1).

Fig. 1—47 year old white male admitted to the hospital with the complaint of abdominal distention following laminectomy done elsewhere six days previously.

Fig. 2—60 year old male admitted to the hospital with the diagnoses of acute suppurative appendicitis. Operated upon, developed peritonitis post-operatively. The scout film of the abdomen shows both large and small bowel dilatation, and the "step-ladder effect" is noted in the small bowel pattern. Upright films showed presence of fluid levels in the loops of bowel.

In the erect position, fluid levels do not necessarily indicate intestinal obstruction, since they are commonly seen in the normal subject in the duodenal bulb, in duodenal and jejunal diverticula, in the flexures of the colon and in haustral pockets of the ascending and transverse colon when its contents are liquid.

In the presence of obstruction the gut is distended, the arrangement of the plicae are characteristic and the fluid levels are longer than in the incidental types mentioned above.[2]

The dictionaries have defined intestinal obstruction as being synonymous to ileus and vice-versa, when actually they are physiologically distinct from one another. Ileus may de defined as a dilatation of the small intestine, or a portion thereof, associated with an accumulation of gas and fluid and with other disturbances in physiology. Obstruction is any localized condition acting as a barrier, or preventing mechanically, the free passage of intestinal contents. Obstruction, usually designated as chronic obstruction, may exist without ileus but ileus often results from obstruction, and is often referred to as acute obstruction. It seems desirable to bear in mind this distinction in terms in order to avoid confusing the two conditions.[3]

In a review of the physiologic disturbances consequent upon ileus, Ochsner (1937)[4] pointed out that distending the intestinal wall results in increased secre-

tion, diminished absorption, and failure to transport intestinal contents from higher to lower levels. The pressure in the wall of the bowel rises and is much higher in the upper than in the lower part of the intestines. The normal function of the duodenum is largely secretory while that of the ileum is largely absorptive, and it has been found that the duodenum secretes fluid five to ten times as rapidly as the ileum. The blood supply of the wall varies in different portions of the intestine. In the upper part the blood vessels enter the wall close to the mesenteric attachment and pass around the circumference deep in the muscle or submucosa. In the ileum, the vessels run part way around just beneath the serosa, sending small branches into the muscle, and then plunge into the wall. Distention of the upper intestine, therefore, compresses the intramural arteries and veins much more than in the lower intestine. Sufficient stretching may obliterate the lumen of the vessels. Storck and Ochsner (1936)[5] summarize the mechanism of interference with intestinal movements by distention as follows: (a) in the early stages it causes fatigue through stretching of the muscles; (b) in the intermediate stages, it causes interference with the blood supply to the muscles, resulting in anoxemia to the bowel; (c) in the terminal stages, it causes actual necrosis of the musculature.

It appears worthy of emphasis that mechanical obstructions must be differentiated from paralytic ileus in cases of distention of the small bowel, because if the diagnosis is not made early by proper roentgenogram studies of the abdomen before the clinical evidence becomes clear, and treatment not started, such obstructions may prove fatal.

Ileus in the early stages may have the appearance of only one hairpin loop, of gas containing intestine, and can be diagnosed long before any clinical evidence becomes clear. As time goes on additional loops become distended, lying side by side producing the so-called "step-ladder effect" (Fig. 2).

The diagnosis of ileus by the demonstration of gas distended loops of small intestine on roentgen films has been done for many years. Hibbard and Swenson showed in dogs that gas in the small intestine can be demonstrated in three to three and one-half hours and that fluid levels appear six to seven hours after an obstruction of the small intestine is produced. The higher the obstruction, the sooner the phenomena developed.[3]

It may be possible to determine to a certain degree the level of gas distended loops by observing the appearance of the mucosal contours visible in the gas shadow. In the high jejunum cross striations can be observed in the shadow (Fig. 2).

In the lower jejunum and upper part of the ileum, these striations become less marked. Lower in the ileum no cross striations can be seen and the margins of the intestine outlined by gas are smooth (Fig. 4).

Consequently, if one observes a smooth loop of intestine the obstruction is probably in the ileum.

The colon when distended by gas presents itself with widely spaced markings, as observed in Fig. 6.

Occasionally, it may become difficult to differentiate distended colon from distended jejunum, but the position of the dilated loop of bowel in the film will aid in distinguishing these portions of intestine (Fig. 3).

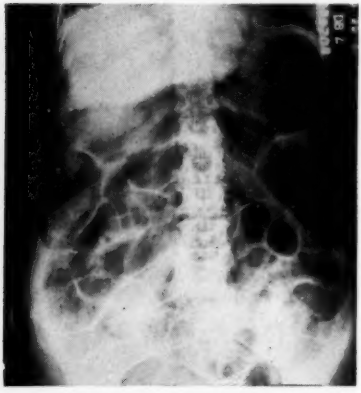

Fig. 3—White, female admitted to the hospital with the diagnosis of obstructive jaundice. Liver biopsy (needle) done 15 days after admission. The following two days the patient began vomiting, complained of pain on the right side of the abdomen which was distended and tympanic. Scout film of the abdomen showed generalized dilatation of the colon and small bowel. The small bowel assumed a so-called "step-ladder pattern." On lateral decubitus projection multiple fluid levels were observed in what appeared to be large and small bowel.

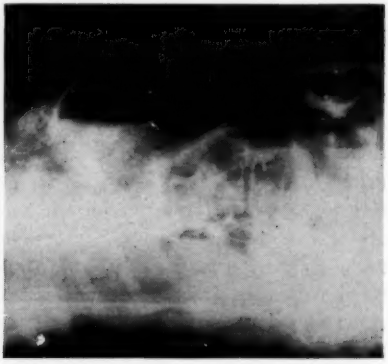

Fig. 4—Same patient described in Fig. 3, on laterial decubitus projection. Note multiple fluid levels seen in what appears to be large and small bowel.

CLASSIFICATION—Ileus may be classified as mechanical, paralytic or adynamic and mixed.

Mechanical Ileus—Mechanical ileus often results from simple occlusion within the bowel, such as a tumor. It may result from simple occlusion from without—adhesions, inflammation, infiltration, or enlarged viscera. Or it may result from incarceration, as observed with adhesions, internal hernia and strangulation. Finally, volvulus is seen in the film as a dilated gas-filled loop of bowel rising out of the pelvis and lying in the middle of the abdomen. This condition can be differentiated roentgenologically from the slowly developing obstruction of the sigmoid due to carcinoma. The distended colon due to gradual obstruction of the sigmoid lies in the usual position and can be traced from the point of obstruction to the cecum. Differentiation of these two conditions is very important to the surgeon.[1]

Ileus may occur proximal to an obstruction when the bowel is no longer able to advance its contents past the critical point, probably initiated by fatigue of the muscle and localized distention, which begins near the obstruction and extends proximally until other portions of the gastro-intestinal tract are involved. Occasionally, the bowel is able to re-establish compensation without mechanical

relief of the obstruction, and is manifested clinically by repeated attacks of abdominal pain and distention with spontaneous relief. The approximate level of the obstruction can be determined by the appearance of the gas distended loops of bowel, as described previously. This has proved of great assistance to the surgeon in planning his operation. The signs presented in a scout film of the abdomen in mechanical ileus are:

(a) hoop shaped gas filled loops of intestine;

(b) fluid levels in motion;

(c) no tumor shadow visible;

(d) little or no gas in colon;

(e) exudate difficult to see;

(f) free diaphragm;

(g) little deformity of adjacent structures;

(h) epiperitoneal fat line not obscured in early films (Fig. 4);

(i) narrowed space between the loops.

Paralytic Ileus—Paralytic or adynamic ileus results from some influence external to the intestine itself which interferes with its physiologic efficiency and starts a train of events similar to those associated with mechanical ileus.[3] The most frequent cause is peritonitis, but other causes such as trauma—injury to the spine, post-operative infections, infections in the lungs and hemorrhage may initiate the train of events. It is important to obtain chest films in cases of abnormal findings in scout films of the abdomen, or try to include the diaphragms in these for subdiaphragmatic free air. Paralytic ileus also results from a disturbance in the circulation of blood in the intestinal wall due to mesenteric vascular occlusion. The differential diagnosis of this from other conditions causing ileus is of great importance and cannot be made from the appearance of the gas shadows on the roentgenogram.[3] Mesenteric vascular occlusion should receive surgical treatment with the least possible delay. This diagnosis is suggested: (a) by a sudden onset of abdominal pain which persists between cramping attacks characteristic of mechanical ileus; (b) by localized tenderness or a palpable tender mass; (c) by fever and leucocytosis. The most important sign is the persistent pain.

Spontaneous paralytic ileus in previously healthy persons is a well known condition in Mysore State, South India, but has received little, if any, recognition elsewhere. W. B. Roantree[5] reports on 33 cases of this rapidly developing paralysis of the small bowel seen during 1936-48. Abdominal X-rays show obstruction with gas and fluid levels. The stepladder arrangement may be found as early as four hours after onset of illness. This early evidence of obstruction speaks against mechanical obstruction, as does the usual absence of increased peristalsis noted in these patients. Pain is usually less severe than in intestinal strangulation, tachycardia and leucocytosis are not seen, and there is no abdominal mass.

Roentgenologically the findings in paralytic ileus may be described as:

(a) meteorism with fluid levels in small bowel and colon, gas in rectum;

(b) signs of inhibition, decreased motor activity;

(c) free peritoneal fluid;

(d) blurring of flank stripe;

(e) inhibition of diaphragmatic excursions;

(f) secondary changes in lung bases, thus the importance of obtaining chest films in these cases; also, for free air under the diaphragms.

(g) epiperitoneal fat line obscured by edema. This is a very important sign when present. If present we may be fairly sure of our diagnosis (Fig. 5).

Mixed Ileus—This usually is a paralytic type followed by a mechanical ileus.

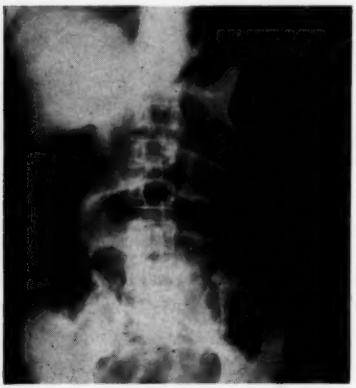

Fig. 5—44 year old colored male admitted to the hospital with acute appendicitis, developed abdominal distention post-operatively. Scout film of the abdomen shows many dilated loops of lower jejunum and possibly upper ileum. Silent abdomen. Note obscured epiperitoneal fat line and presence of exsudate.

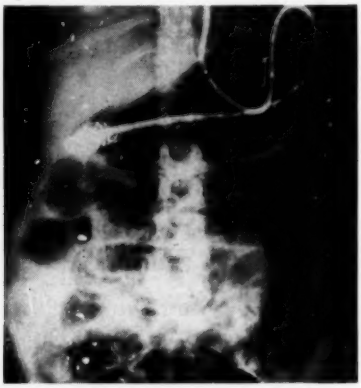

Fig. 6—There is an intestinal drainage tube coiled in the stomach with the mercury filled bag lying just proximal to the pylorus. Below this are several dilated loops of jejunum, actually the dilation goes down as far as the upper ileum. There is a small amount of gas in the colon, but the colon here is not a pattern of obstruction. Clinical findings with scout films of the abdomen suggest paralytic type of ileus.

DIFFERENTIAL DIAGNOSIS—It may be very difficult to differentiate roentgenologically mechanical from paralytic ileus. The clinical history is very important to help in evaluating the case. Haworth and Garland (1940)[6] have pointed out that in the late stages of mechanical ileus, the differential diagnosis from the paralytic type is usually very difficult because by that time gas begins to accumulate in the large intestine. The properitoneal fat line (of diminished density), visible along the flank, becomes partially obscured by edema in the presence of peritonitis, but they have found it also obscured in the late stages of mechanical ileus.

Generally, gas is not observed in the large bowel in mechanical ileus except in very small amounts in adults, whereas in paralytic ileus gas can be observed in considerable amount in the colon, rectum and stomach (Fig. 6). Fluid may also be observed in the colon. In paralytic ileus, there is inhibition of the bowel, and

fluid levels may be observed at the same height[7] (Fig. 1). Changes in the lung bases are also more common in adynamic ileus, and there may be free sub-diaphragmatic air.

In paralytic ileus with free fluid in the abdomen, the X-ray has a characteristic appearance when taken with the patient in the upright position. The gas distended coils rise to the upper part of the abdomen, while the lower abdomen appears opaque as a result of the effusion (Fig. 1).

Extreme meteorism dominates the picture in paralytic ileus.

There are occasions in which differential diagnosis between mechanical, spastic and inhibitive (paralytic) ileus may be difficult or impossible to make. The consideration which causes the clinician the greatest concern is whether a strangulating mechanical obstruction may be present. Wangensteen[8] has encountered this dilemma many times, once in a severe post-operative wound infection which gave very few local signs.

SUMMARY

The scout film of the abdomen is a practicable and valuable diagnostic procedure, as an aide to the diagnosis of ileus.

In suspected small bowel obstruction the diagnosis should be made early by proper roentgenogram studies of the abdomen before the clinical evidence becomes clear, because if treatment is not started such obstructions may prove fatal to the patient.

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SUBACUTE ENTEROCOCCAL ENDOCARDITIS FOLLOWING EPIDEMIC HEMORRHAGIC FEVER

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Since the Korean war American physicians have become acquainted with *epidemic hemorrhage fever*.[1] Despite intensive study many of its basic features continue to defy definition. The etiological agent remains unknown. Sequelae and recrudescenses have not been reported.

Recently, we treated a young man with subacute bacterial endocarditis who previously had epidemic hemorrhagic fever. The case is reported to present a possible sequel of epidemic hemorrhagic fever not heretofore described, and to suggest that this sequel be kept in mind when treating returning Korean war veterans.

REPORT OF A CASE

A 22 year old white male tool crib attendant presented himself at the Henry Ford Hospital Emergency Room, May 22, 1953, complaining of chills and fever of 12 hours duration. For three days he had noted asthenia and mild malaise. The day of admission he developed a severe frontal headache, sore throat, and dry hacking cough, followed by backache, urinary frequency, urgency, and dysuria.

In September 1951 while with the army in Korea, 75 miles north of Seoul, he contracted epidemic hemorrhagic fever. His symptons were identical to those of the present illness, except that in addition he had had gross hematuria. From September to December of 1951, he was treated in an army hospital in Japan. Following this hospitalization, he remained "weak" until the summer of 1952. By August he felt well enough to work regularly until the present illness.

Except for the hemorrhagic fever, he had enjoyed good health while in the army. He took chloroquine regularly as prescribed in Korea and primaquine on the ship enroute back to the United States.

The patient had never had rheumatic fever and denied ever having had joint pains, epistaxis, chorea, or "growing pains." Neither at the time of his army physical examinations, nor at any other time had he ever been noted to have any evidence of heart disease.

On admission May 22, 1953, the physical examination revealed the following: He appeared acutely ill, with an oral temperature of 103° F., and was shaking with a chill. The conjunctivae were markedly injected, and there was photophobia. Funduscopic was normal. The pharynx was intensely inflamed and exhibited shallow ulcerations. The gums were spongy, swollen, and bleeding. The abdomen was diffusely tender. The patient appeared nauseated.

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The blood pressure was 125/80 mm. of Hg. Pulse was 124 per minute. In the aortic area there was a soft to-and-fro pericardial friction rub. There were no murmurs. The lungs were resonant and no rales were heard; breath sounds were slightly harsh but well transmitted. There were no petechiae and all joints were freely mobile and non-tender. There was no significant lymphadenopathy. The remainder of the physical examination was not remarkable.

Hospital Course—A venous blood culture drawn the day of admission yielded an enterococcus.

The patient remained febrile for two days. With the preliminary report of the positive blood culture on the third hospital day, intramuscular injections of streptomycin (½ gram) and penicillin (400,000 units) twice daily were begun. The temperature promptly fell to normal by lysis the next three days.

Substernal pain, worse on coughing, relieved by sitting up and leaning forward, persisted for over one week, but the pericardial friction rub was no longer audible. Electrocardiograms taken the second hospital day and periodically thereafter were characteristic of pericarditis.

On the fourth hospital day two splinter hemorrhages were detected beneath the fingernails. That day, a soft systolic aortic murmur was audible for the first time. This murmur increased in intensity to a maximum the third hospital week; it subsided somewhat then, but remained distinctly present.

When complete studies of the organism causing the bacteremia proved that it was an enterococcus, antibiotic therapy was intensified. On the twelfth hospital day an indwelling intravenous polyethylene catheter was inserted, and a continuous infusion of 5% glucose in water was begun, via which 20 million units of penicillin were administered daily. The intravenous solution also contained 50 mg. heparin sodium per 1000 cc., a dosage which in our experience will obviate intra-catheter clotting without producing detectable systemic anticoagulation.[2] One-half gram of streptomycin plus one-half gram of dihydro-streptomycin were injected intramuscularly twice daily.

Although the patient had remained afebrile, on the eighteenth hospital day he suddenly developed acute generalized abdominal pain and vomited some material containing a small amount of fresh blood. A scout film of the abdomen revealed a paralytic ileus. Several large petechiae appeared on the soft palate and buccal mucosa, as well as some new ones in the nailbeds. One of the petechiae of the nailbed was removed and examined microscopically; numerous pleomorphic short-chained cocci were observed. Blood cultures obtained at this time remained sterile. Conservative treatment with Miller-Abbott tube drainage, hot stupes, and antispasmodics was successful.

On the twenty-fifth hospital day, dental x-rays revealed five devitalized and infected teeth, as well as two retained root fragments. These teeth and root fragments were removed at two sittings. Blood cultures at the time of extraction proved sterile. The teeth were cultured and yielded Candida albicans, Staphylococcus albus (coagulase negative), Aerobacter aerogenes, and Escherichia coli.

Except for two days of temperature up to 100° F at the time of the abdominal crisis, the patient had remained afebrile since the first few days after admission. On June 25 the antibiotics were discontinued, and on June 27, the 35th day of hospitalization, he was discharged well.

Laboratory Data—The day after admission the red cell count was 5.19 million per cu.mm., hemoglobin 14.5 grams per cent, white cell count 10,900 per cu.mm. with 75% neutrophiles and 25% lymphocytes. Counts were obtained daily thereafter for one week and then three times a week until discharge. Except for some neutrophilia at the time of the abdominal crisis (12,650 white cells per cu.mm. with 81% neutrophiles), the counts were remarkable only in that there was usually a slight monocytosis (average 10%) and eosinophilia (average 3%). There were no leucocytoid lymphocytes and no malaria parasites.

Erythrocytic sedimentation rates were obtained semi-weekly and ranged from 5 to 15 mm. per hr. (Wintrobe, corrected.) Hematocrit ranged from 48 to 54 mm. Three platelet counts were made the first week after admission and were 246,000, 228,000 and 368,000 per cu.mm. Bleeding and clotting times and tourniquet tests were normal.

Urinalysis was done daily. Traces of albumin were present the ninth and seventeenth hospital days; granular casts were present those days and also on the twenty-eighth hospital day. There was never any hematuria or pyuria. From the 13th to the 16th hospital days, all voided urine specimens were checked for albumin; one of these contained three plus albumin, but all others were negative.

Fasting blood sugar, blood cholesterol, and frequent blood non-protein nitrogen determinations, were normal. The Kline exclusion test was negative.

Agglutinations for typhoid, paratyphoid A and B, Brucella abortus, Proteus OXK, OX2 and OX19 were done on the 2nd, 10th, and 31st hospital days. All were negative except the proteus OX19, which exhibited titers of 1:80 and 1:40 the first two tests, but was negative the third time. Weil's agglutination tests done on the 10th and 31st hospital days were negative. Heterophile agglutinations on the 2nd and 31st hospital days were negative. Antistreptolysin 0 titers on the 2nd, 10th, 18th and 31st hospital days showed no significant rise.

In addition to the first blood culture, which was positive, nine others taken at four to six day intervals were negative. Throat culture yielded, beta, hemolytic streptococcus, Hemophilus influenza, and Neiseria catarrhalis. Two urine cultures, two stool cultures, and two warm stage stool examinations were negative.

Urea clearance test demonstrated normal clearance. Phenolsulphonphthalein excretion was 65% the first hour. Intravenous pyelograms were negative. Chest x-ray was normal.

Prothrombin time was 13 seconds, 100% of normal. Cephalin cholesterol, thymol turbidity, and thymol flocculation were done the 3rd and 13th hospital days and were normal. Van den Bergh determination revealed a total of 0.77 and direct of 0.19 mg. per cent. Icterus index was 4 units. Serum albumin was 4.1 grams per cent and serum globulin was 2.1 grams per cent.

Case Comment—The diagnosis of subacute bacterial endocarditis was established by the demonstration of positive blood culture (enterococcus), bacterial emboli, development of a murmur, and presence of fever and chills. The diagnosis of pericarditis was established by the presence of a pericardial friction rub, substernal pain aggravated by coughing and deep breathing and relieved by sitting forward, and by the presence of typical electrocardiographic changes.

DISCUSSION

Two features of this case are of particular interest. The first is the nidus upon which bacterial endocarditis developed; the second is the etiology of the pericarditis.

Bacterial endocarditis rarely involves a normal valve and when it does, the clinical course and bacteriology are characteristic. The simultaneous onset of subacute bacterial endocarditis and acute rheumatic endocarditis has been reported[3] but is exceedingly uncommon. If the endocardium, valvular or otherwise, was damaged in this patient prior to his present illness, three possibilities may be considered: Congenitally deformed valves or septa are sometimes involved by bacterial endocarditis, but these are almost always detectable by auscultation. Previous valvulitis, for example from rheumatic fever, also should be detectable clinically, but in addition, there was no history of any form of rheumatic fever. The sedimentation rate was not elevated on admission, the antistreptolysin titer did not rise significantly, and the electrocardiogram did not reveal any delay in the auriculoventricular conduction time.

Another possible explanation for the valvular or other endocardial damage is the previous epidemic hemorrhagic fever. Although no valvulitis per se was described in the autopsies recently reviewed,[1d] all these were in patients in the acute phase of the disease. Subendocardial hemorrhages are a hallmark of the disease and all four chambers as well as the aorta itself have been involved.[1e] It then seems logical to deduce that slight residual roughening of the endocardium not detectable by auscultation, may follow epidemic hemorrhagic fever. We consider this the most tenable explanation in this case.

Pericarditis may be associated with subacute bacterial endocarditis. It is not uncommon in acute rheumatic fever. It also occurs following upper respiratory infections. All three of these possibilities can be considered in this case. A fourth consideration, however, is introduced by the fact that subepicardial hemorrhages have been found in a number of cases of epidemic hemorrhagic fever.[1e] In this case it is difficult to assign the etiology of the pericarditis. The similarity of the symptoms, at the onset, to those which ushered in the hemorrhagic fever, i.e., the presence of tracheobronchitis, gastritis, pharyngitis, rhinitis, conjunctivitis, and possible peritonitis, as well as dysuria and frequency, suggest the possibility that the patient's epidemic hemorrhagic fever residuals, if not the disease itself, underwent a transient recrudescence.

Intermittent albuminuria and the cylindruria have been described as occurring in epidemic hemorrhagic fever.[1] These also occur as a consequence of renal emboli

during the course of subacute bacterial endocarditis. In view of the history of gross hematuria with the Korean illness, and because of the marked urinary tract symptoms despite minimal laboratory and physical findings, it is possible two pathological processes existed. Even though there were probably some renal emboli, the early urinary symptoms may be the result of acute toxicity at the onset of the illness aggravating damage in the kidneys residual from the epidemic hemorrhagic fever.

Teleologically we have reconstructed the case as follows. The diagnosis of epidemic hemorrhagic fever in Korea is accepted. Poor oral hygiene produced a focus from which enterococcemia arose.[2] Subacute bacterial endocarditis developed in the region of the aortic valve roughened by the epidemic hemorrhagic fever. When the subacute bacterial endocarditis was adequately treated, the entire illness subsided.

SUMMARY AND CONCLUSIONS

1. A case of subacute enterococcal endocarditis occurring in a patient who had had epidemic hemorrhagic fever is described.

2. Reasons for concluding that the endocarditis involved endocardium previously roughened by epidemic hemorrhagic fever are presented.

3. It is suggested that this possibility be kept in mind when treating patients who have had epidemic hemorrhagic fever.

4. The validity of these conclusions will depend on long-range follow up of other patients who have had epidemic hemorrhagic fever.

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THE HENRY FORD HOSPITAL MEDICAL SOCIETY PROGRAM FOR FEBRUARY TO APRIL 1954

William E. Jahsman, M.D., *President* C. Paul Hodgkinson, M.D., *Vice President*
John W. Sigler, M.D., *Secretary*

February 9, 1954—8:15 p.m.—Henry Ford Hospital Auditorium

Roentgen Manifestations of Acute Abdominal Disease

William R. Eyler, M.D., Associate Physician, Department of Radiology, Henry Ford Hospital

March 9, 1954—8:15 p.m.—Henry Ford Hospital Auditorium

Some Problems of Biliary Tract Disease

I. S. Ravdin, M.D., Professor of Surgery, University of Pennsylvania, Philadelphia, Pennsylvania

April 13, 1954—8:15 p.m.—Henry Ford Hospital Auditorium

The Certified Chaplain and the Modern Hospital

Russell L. Dicks, Ph.D., Litt. D., Professor of Pastoral Care Divinity School, Chaplain of Duke Hospital, Durham, N. C.

April 30, 1954—8:15 p.m.—Henry Ford Hospital Auditorium

The Roy D. McClure Memorial Lecture

The Expanding Scope of Cardiovascular Surgery

Alfred Blalock, M.D., Surgeon-in-Chief, Johns Hopkins Hospital, Baltimore, Maryland

ABSTRACTS OF RECENT PUBLICATIONS OF THE PROFESSIONAL STAFF OF THE HENRY FORD HOSPITAL AND THE EDSEL B. FORD INSTITUTE FOR MEDICAL RESEARCH

NUTRITIONAL STUDIES IN ADOLESCENT GIRLS, AND THEIR RELATION TO TUBERCULOSIS. Joseph A. Johnston, Springfield, Ill. C. C. Thomas, 1953.

This book is an impressive record of a twenty year nutritional and metabolic study on adolescent girls, most of whom had tuberculosis. It gives in detail the evidence to show that the abrupt increase in the adult form of tuberculosis in adolescence is related to a failure to meet the nutritional requirements for growth peculiar to this period. Evidence is given also for the belief that a favorable course of the disease process, may be conditioned by a normal nutritional state and replenishing previous deficits. Here are brought together also the author's studies with balance data of the food requirement in the adolescent, and the various factors which influence its utilization. Among these factors are included, the effect of thyroid on nitrogen and calcium retention, the effects of rest and activity, the requirements of vitamins, the contrasting metabolic state in the pre- and post-pubertal periods, the effect of the hormones, pituitary, ACTH, estrogen, androgen and adrenal, and the effect of focalinfection.

Suggestive observations have appeared, for example, when failure to meet the food requirement resulted in inadequate retention with a presumably adverse effect on the disease process. Within limitations these studies show a state of nitrogen and calcium depletion associated with disease, positive healing associated with positive nitrogen balance, a positive calcium balance to be a protection against primary focus progression, and that a variety of factors, of which diet is one, play a role in the management of the adolescent. The material reported here has been a vital contribution in pediatric disease.

SODIUM AMYTAL NARCOSIS, WITH DELIRIUM, AS A THERAPEUTIC METHOD. Eugene J. Alexander. Dis. Nerv. System 14:195, 1953.

Experience is reported in the treatment of 324 cases by prolonged narcosis from Sodium Amytal, followed by delirium as a result of abrupt discontinuance of the drug. Although the psychoneurotic patients were, on the whole, persons who had been seriously disabled for a long time, and had not responded to psychotherapy, from 57 to 60 percent (of 205) gained significant improvement.

Since only 40 (34 percent) cases gained improvement out of 119 treated for conditions other than psychoneurosis, the treatment is not now recommended unless there is a significant psychoneurotic component in the patient's illness.

Six patients (of 343 begun on treatment, i.e., 1.5 percent) died, but from what we have learned we are sure this figure can be reduced. Moderately severe complications occurred in 9 percent of cases, but teamwork and experience with the method can also reduce that figure.

Several theories are examined as to the mode of action of the therapy; and the one which appeals is that the amytal-delirium treatment provides a psychological setting in which some basic neurotic fears can be faced and lived through, even without verbalization.

Many patients after Sodium Amytal therapy are more accessible to psychotherapy; and the improvement rate can probably be definitely increased by properly timed insight psychotherapy.

PRESENT STATUS OF SURGICAL PROCEDURES FOR URETERO-ENTERIC ANASTOMOSIS. A. Waite Bohne. J. Urol. 70:581, 1953.

Eleven basic surgical principles from the literature are enumerated and related to this highly specialized operation. All eleven principles were developed to overcome the basic hazards, leakage at the site of anastomosis, obstruction of the urinary tract, and infection of the urinary tract. Preliminary permanent colostomy would seemingly offer the most effective way to overcome the hazard of infection, the only disadvantage being the social burden of an inguinal colostomy. Obstruction of the urinary tract must be eliminated by the actual transplantation technique. Only the direct method offers such freedom. Leakage at the site of anastomosis may be controlled by proper placement of sutures, and the leaving of a strip of parietal peritoneum attached to the ureter, to allow rapid sealing of the anastomotic site. The hazard of electrolyte balance concerning which 80 percent of these patients manifest some degree of acidosis, may be controlled by detailed attention to gastro-intestinal absorption.

FINGER TIP INJURIES. Robert H. Clifford. S. Clin. North America 33:1311, 1953.

Finger tip injuries are considered as those injuries affecting tissues distal to the distal interphalangeal joint. In treatment, local anesthesia is usually advised. For soft tissue losses exposing bone or involving most of the finger pad it is best to use a pedicle flap. Compound fractures can be reduced without difficulty. The finger nail base can often be reinserted in its normal bed. In cases of clean amputation choice of treatment lies between revision and closure, and a cross finger or cross palm flap. For the index finger, the choice of donor site for cross finger flap is the dorsal skin of the middle phalanx of the middle finger. For the thumb, the source of tissue can be either the dorsum of the proximal phalanx of the index finger or the distal portion of the palm over the second metacarpal head. Finger tip injuries are important, and treatment must consider the occupation of the patient, and be planned in detail with this in view.

PANCREATIC CYSTS. Laurence S. Fallis and James Barron. Am. J. Surg. 86:255, 1953.

Pancreatic cysts, like other disorders of the pancreas, are of relatively rare occurrence; therefore, no one surgeon is in a position to consider the problem on the basis of his own personal experience. The collected experience of groups of surgeons is also meager, thus accounting for variation in treatment, and disparity in results. True pancreatic cyst may be congenital, retention, degenerative or parasitic in type. False or pseudocysts may be traumatic or inflammatory. Surgical intervention when a cyst is present is mandatory and may be of four types, aspiration, excision, external drainage marsupialization, and internal drainage—enteroanastomosis. Every cyst wall should be biopsied.

BODY POSTURE AND BLOOD PRESSURE. C. Paul Hodgkinson and Robert C. Rood. Obst. & Gynec. 2:399, 1953.

A detailed presentation is given here of the average ovarian vein blood pressures in three Groups of patients undergoing gynecologic operations. In Group I, the operating table was horizontal, in Group II the patient's head was raised 15° above horizontal and in Group III the patient's head was lowered from horizontal 15°. The venous pressures in Group I averaged 14.82 cm water, in Group II it averaged 19.66 cm water, and in Group III it averaged 8.18 cm water. In addition, a review is given of the influence of body posture upon arterial and venous blood pressure in gynecologic surgery.

THE INFLUENCE OF BODY POSTURE UPON ARTERIAL AND VENOUS BLOOD PRESSURE IN GYNECOLOGIC SURGERY, by C. Paul Hodgkinson and Robert C. Rood. J. Am. Nurse Anesthetists 21:157, 1953.

The influence of body posture on circulation should be applied to everyday surgical problems. The head-down position for acute hypotension, and the head elevated position for impaired cardiac function are recognized treatments. Clinical experience with gynecological patients suggested that alterations in body position during operation are more important than generally appreciated. Three groups of patients were studied. Group I patients were kept in 15 degree Trendelenburg position until they reacted from anesthetic, and then were returned to horizontal gradually. There was no fall in arterial blood pressure. Group II patients were abruptly placed in horizontal position from 15 degree Trendelenburg, after operation, and 2 of 10 required drug treatment and transfusion for hypotension. Group III patients, after operation in 15 degree Trendelenburg position, were abruptly changed to 15 degree head-upward position. The average blood pressures decreased from 128 mm. Hg systolic, and 65 mm Hg diastolic to 68 mm Hg systolic, and 46 mm Hg diastolic. Most patients were returned at once to the Trendelenburg position. In hypotension the head-down position favors mobilization of the blood of the lower extremities, which is normally estimated at 550cc. Upon termination of an operation tilting the patient abruptly upwards from the Trendelen-

burg position may deprive the circulation of a like amount of blood. Gradual changes in patient position, and the use of elastic bandages are important aids in maintaining circulating blood volume.

ROENTGEN STUDY OF URETHROVESICAL RELATIONSHIPS IN FEMALE URINARY STRESS INCONTINENCE. C. Paul Hodgkinson and Howard P. Doub. Radiology 61:335, 1953.

An abstract from the Am. J. Obst. & Gynec. 65:560, 1953 embodying substantially this same material was published in the Henry Ford Hospital Medical Bulletin 1:43 (Sept.) 1953.

QUANTITATIVE VARIATIONS IN THE RESPIRATION OF THE NEWBORN INFANT. Philip J. Howard and A. Robert Bauer. Am. J. Dis. Child. 86:284,1953.

Additional observations have been made on the respiration of 14 sleeping newborn infants ranging in age from 8 days to 46 days in an effort to re-examine the trend and range of normal with reference to minute volume, tidal air, rate and pattern. Special attention was given to the 20th to 30th minute of sleep to establish, if possible, basal averages. It was suggested that the lowest minute volume observed, without regard to how much sleep preceded or followed it, might, in fact, be the best indication of basal condition. Respiration is of multiple patterns; its component parts rate, and tidal air have a wide range, and the minute volume is variable even within sleep. The authors compare their results with those of other observers in this field.

THE MECHANISM OF DEATH FROM INTRACARDIAC AIR AND ITS REVERSIBILITY. Thomas Geoghegan and Conrad Lam. Ann. Surg. 138:351, 1953.

An abstract from the Am. Heart J. 46:215, 1953 embodying substantially this same material is published in this issue of Henry Ford Hospital Medical Bulletin.

ELECTROCARDIOGRAPHIC MANIFESTATIONS OF AIR IN THE CORONARY ARTERIES OF DYING AND RESUSCITATED HEARTS. Thomas N. James, Thomas Geoghegan and Conrad R. Lam. Am. Heart J. 46:215, 1953.

The fate and consequences of intravascular air form a subject which has attracted medical attention and investigation since before 1830, when Cormack blew "the contents of his chest, twice filled" into the veins of a horse. Within the last three decades the vast difference in the effect of air in the right side of the heart as compared to that in the left side has become apparent. Excepting the presence of shunts, venous air in small amounts is relatively innocuous. Although this is not true when a gas enters the left side of the heart, until recently the reason has been controversial. Present evidence favors occlusion of the coronary arteries as the mechanism by which death occurs in the latter example.

Electrocardiograms taken during coronary artery occlusion by air are rare. Nine adult dogs were used in this investigation and 1.5cc of air per kilogram was injected rapidly near the mid portion of the left ventricle. Observations were made of the electrical and mechanical dissociation in the dying heart; of the electrocardiogram of the resuscitated heart; of the auricular complexes; of conduction disturbances; and of aortic pressure.

Injection of lethal amounts of air into the left ventricular cavity is followed by occlusion of the coronary arteries by this air. The electrocardiogram initially manifests an injury current following this occlusion but soon reverts to a non-pathologic contour. During this phase there is gross dissociation between the mechanical and electrical activity of the heart. As the anoxia progresses, various conduction disturbances appear. Ventricular fibrillation is uncommon.

The changes produced by air in the coronary arteries are entirely reversible, with the recovered animal exhibiting no electrocardiographic evidence of residual injury to the heart.

CRYOGLOBULINS IN CORONARY-ARTERY DISEASE. Thomas N. James and Ellet H. Drake. New England J. Med. 249:601, 1953.

Cold-precipitable substances in serum have been termed "cryoglobulins." This substance when present in the blood serum in large amounts produces a clinical syndrome similar to Raynauds disease. Since large amounts of cryoglobulin cause the blood to gel, the present speculation is the question: "Do small amounts increase blood viscosity to a lesser degree, but prove hazardous to patients with narrowed blood vessels?" Tests were devised for assaying the traces of cryoglobulin in blood specimens at 5° C. and 37° C. The viscosity of serum, from negative and positive patients, the former showing no cryoglobulin, the latter showing cryoglobulin present, was unchanged at 5° C. and 37° C. The serum viscosity, therefor, was unaffected by this amount of cryoglobulin. In view of the normal viscosity, the significance of the small amounts of cold precipitable substances present in 40.6 percent of 69 patients, is left open to speculation. No correlation was found between cryoglobulinemia and myocardial infarction in 65 cases.

CARCINOMA OF THE CERVICAL ESOPHAGUS AND LARYNX; TREATMENT OF AN ELDERLY PATIENT BY ESOPHAGOLARYNGECTOMY (Wookey Operation). Conrad R. Lam and E. J. Gordon. J. A. M. A. 153:552, 1953.

It is well known that some malignant tumors, especially in elderly persons, grow slowly, and, if they are excised, the prospects for cure are excellent. This case is reported because it illustrates the difficulty in the early diagnosis of lesions that produce dysphagia and the possibilities of cure by operation in carcinoma of the cervical esophagus even after the symptoms have been present for many months and after the larynx has become involved. A woman of 78 years with a history of difficulty in swallowing over a period of more than two years was found to have

carcinoma of the cervical esophagus with involvement of the larynx. Surgical treatment by the method of Wookey (cervical exophagolaryngectomy with cutaneous esophagoplasty and permanent tracheostomy) gave gratifying results. The more frequent and early use of the esophagoscope in the diagnosis of lesions that produce dysphagia is suggested.

COMBINED MITRAL AND PULMONARY ATRESIA. Conrad R. Lam, E. M. Knights and R. F. Ziegler. Am. Heart J. 46:314, 1953.

Congenital mitral atresia is itself a rare anomaly and its combination with pulmonary atresia has not been reported previously. The possibility of an infant surviving the neonatal period with no clear-cut way of blood reaching the lungs or leaving them would have seemed extremely remote. Nevertheless, this child was able to exist for three months, at which time operative intervention to relieve the cyanosis was attempted. This infant of three months, a complete description of whom is included, died following a Potts operation (anastomosis between the aorta and the left pulmonary artery). Autopsy showed a hitherto undescribed anomalous structure of the heart. There was atresia of both the mitral and pulmonary valves, together with a questionably patent ductus arteriosus, intact interatrial septum, and an interventricular septal defect.

RESPIRATORY TRACT ABSORPTION OF CRYSTALLINE B_{12} IN MAN DEMONSTRATED BY URINARY BIOASSAY AND HEMATOPOIETIC STUDIES. R. W. Monto. Fed. Proc. 12:396, 1953.

An article embodying substantially this material was published in the Henry Ford Hospital Medical Bulletin 1:15 (March) 1953.

MEGALO-URETER AND RELATED CONDITIONS IN CHILDREN: REPORT OF SIX CASES. John K. Ormond. J. Urol. 70:171, 1953.

Six cases of ureteral dilatation in children are reported in detail and their operative treatment. Some conclusions seem justified. Reflux of the ureters is not amenable to operative treatment. It seems probable that removal of part of the ureter to remedy tortuosity and kinking resulting from long continued reflux may be useful. Excision of both ends of the ureter with re-establishment of continuity does not result in harm. The work of Svenson and his associates makes it very probable that in the past efforts to remedy or palliate megalo-ureter have been misdirected; that the bladder is the seat of the trouble and not the ureter. Up to date no means of permanently remedying the condition known as megalo-ureter has been devised.

ZINC PHOSPHATE IDENTIFIED AS A CONSTITUENT OF URINARY CALCULI. Jonathan Parsons. Science 118:217, 1953.*

For several years x-ray diffraction has been used in the physics department of this institute for the analysis of urinary calculi. During this time 192 stones have

*From Edsel B. Ford Institute for Medical Research.

been studied. In March 1951 a calculus was received 39 mm long, which analyzed as principally carbonate and magnesium ammonium phosphate, but contained yellowish layers of material of different pattern. Spectrographic analysis of this indicated zinc and phosphorus as the major elements. Diffraction spacings for $Zn_3 (PO_4)_2 . 4H_2O$ agreed closely with the pattern lines. Zinc is known to be a constituent of most foods, and although small traces occur in the urine, most is excreted through the intestinal tract.

METALLIC GRATING REPLICAS AS INTERNAL STANDARDS FOR CALIBRATING ELECTRON MICROSCOPES. S. R. Rouze and John H. L. Watson, J. App. Physics 24:1106, 1953.*

It was shown earlier that plastic grating-replicas mounted upon eighth-inch specimen screens were practical internal standards and that they could be accurately calibrated independently by spectroscopic means. Metallic replicas of diffraction gratings have been made by a direct-stripped method. These can still be direct-stripped easily and without damage to the original grating, and the optical quality of these pre-shadowed all-metal replicas is better than that of the plastic replicas. Because of their superior optical properties, the spacing can be determined spectroscopically more conveniently and with greater accuracy. In addition, the metal-gratings are more stable than the plastic ones. The electron microscopy of the metallic replicas and the accuracy and constancy of their magnification measurement is discussed.

*Research Laboratories Division, General Motors Corporation and Edsel B. Ford Institute for Medical Research.

GOUTY ARTHRITIS. John W. Sigler and Dwight C. Ensign. J. Michigan M. Soc. 52:959, 1953.

Gout is still a relatively common though frequently overlooked cause of acute and chronic joint disease. Although the fundamental metabolic disturbance is poorly understood and so far cannot be corrected, there are effective methods of controlling acute symptoms. These methods are rest, local protection, and moist compresses, a purine-free diet, acetylsalicylic acid, and colchicine, with attention to detail and with persistence. A patient with chronic tophaceous gout needs careful renal function evaluation. Refractory chronic patients may prove to need corticotropin, hydrocortisone intraarticularly, cortisone orally, or the more toxic phenylbutazone. By simple but prolonged and faithfully followed treatment, the incidence of acute attacks can be diminished, and chronic attacks delayed or prevented.

THE USE OF NEBULIZED TRYPSIN; A PRELIMINARY REPORT. J. Lewis Yates and Ben E. Goodrich. Dis. Chest 24:320, 1953.

Viscid bronchial secretions and exudates are important causes of disability in respiratory disease. Liquefaction by nebulised proteolytic enzymes seemed a

logical approach to this problem. A solution containing 40,000 to 50,000 Armour Units of crystalline trypsin in 1cc. of Sorensen's phosphate buffer solution was nebulised by 100 percent oxygen, and inhaled three to six times daily, an average course being 5 inhalations of 1cc. each. The viscosity of the sputum was decreased. The volume of sputum increased at first, and later decreased. Thirteen of seventeen patients improved as noted by decreased dyspnoea, wheezing and rales. The only untoward reactions were mild local irritation of the pharyngeal mucosa. Suggested indications are bronchiectasis, bronchitis, post operative atelectasis, and as an adjunct to local examination of the bronchi.

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Tube feeding (Barron) ..... June, *13*
Tuberculosis. Congenital (Kempton & Howard) ..... Sept., *6*
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